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TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE TWENTY-FIRST.

MAR 23 1900

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1899.

NOTICE.

The present volume of TRANSACTIONS contains the papers read before the College from January, 1899, to December, 1899, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

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1899.

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COMMITTEE ON THE NATHAN LEWIS HATFIELD PRIZE (Until Feb. 1901).

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LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES *
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS †
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.

* Died four months after his election.
† Resigned on account of ill-health.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1899.

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

*1883. Abbot, Griffith E., Ph.D., M.D., Washington, D. C.

1892. Abbott, Alex. C., M.D., Professor of Hygiene and Bacteriology, and Director of the Laboratory of Hygiene in the University of Pennsylvania.

1876. Alison, Robert H., M.D.

1873. Allis, Oscar H., M.D., Surgeon to the Presbyterian Hospital.

1896. Allyn, Herman B., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Visiting Physician to St. Joseph's Hospital; Medical Registrar to the Philadelphia Hospital.

1888. Anders, James M., M.D., LL D., Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical and Samaritan Hospitals.

1869. Andrews, T. Hollingsworth, M.D., Medical Director of the Bureaus of Police and Fire, and Commandant of the Philadelphia Emergency Corps of the Department of Public Safety.

1896. Angney, William M., M.D., Physician to the House of Mercy (Home for Male Consumptives); Consulting Physician to the Hospital for Diseases of the Lungs at Chestnut Hill.

*1882. Ashbridge, Richard, M.D., West Whiteland, Pa.

ELECTED

1863. Ashhurst, John, Jr., M.D., LL.D., Barton Professor of Surgery and Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania Hospital and to the Children's Hospital; Consulting Surgeon to St. Christopher's Hospital, to the Woman's Hospital and to the Hospital of the Good Shepherd, Radnor.

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1893. Ashton, Thomas G., M.D., Physician to the Philadelphia Hospital; Assistant Physician to the Jefferson Medical College Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania.

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

1852. Bache, Thomas Hewson, M.D.

1883. Baer, Benjamin F., M.D., Professor of Gynecology in the Philadelphia Polyclinic.

†1892. Baker, George Fales, B.S., M.D.

1879. Baker, Washington H., M.D., Obstetrician to the Maternity Hospital.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital and to the Gynecological Out-patient Department of the Pennsylvania Hospital; Consulting Surgeon to the Frederick Douglass Memorial Hospital.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home.

1880. Bartholow, Roberts, M.D., Professor (Emeritus) of Materia Medica, General Therapeutics and Hygiene in the Jefferson Medical College.

1894. Barton, James M., M.D., Surgeon to the Jefferson Medical College Hospital and to the Philadelphia Hospital.

1883. Baum, Charles, A.M., M.D., Ph.D.

1883. Beates, Henry, M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician-in-Charge to the Seashore

ELECTED

Home for Invalid Children, and to the Seaside House for Invalid Women, Atlantic City; Formerly Physician to the Episcopal Hospital, and Physician-in-Charge to St. Christopher's Hospital for Children.

1896. Beyea, Henry D., M.D., Instructor in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital.

†1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

*1866. Black, J. J., M.D., New Castle, Del.

1894. Bliss, Arthur Ames, M.D., Laryngologist and Aurist to the German Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

*1867. Boardman, Charles H., M.D., Evanston, Illinois.

1894. Bochroch, Max H., Instructor in Electro-therapeutics and Chief Clinical Assistant in the Nervous Department of the Jefferson Medical College Hospital; Neurologist to the Outpatient Department of St. Joseph's Hospital.

1896. Boger, John A., M.D., Surgeon to St. Mary's and the Samaritan Hospitals; Surgeon to the Dispensary of the Episcopal Hospital.

1891. Boyd, George M., M.D., Clinical Professor of Obstetrics in the Medico-Chirurgical College; Physician to the Philadelphia Lying-in Charity Hospital and Nurse School.

†1884. Bradford, T. Hewson, M.D., Medical Director of the United Security Life Insurance and Trust Company of Pennsylvania; Medical Examiner of the Equitable Life Assurance Society of the United States and of the John Hancock Mutual Life Insurance Company, Boston, Mass.

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College; Consulting Surgeon to St. Joseph's Hospital and to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D., Visiting Physician to the Nervous Department of the Howard Hospital.

1887. Brubaker, Albert P., M.D., Professor of Physiology in the Pennsylvania College of Dental Surgery; Adjunct Professor of Physiology and Hygiene in the Jefferson Medical College; Lecturer on Anatomy and Physiology in the Drexel Institute.

ELECTED

*1890. Brush, Edward N., M.D., Medical Superintendent of the Shepherd and Enoch Pratt Hospital, Towson, Md.

*1851. Bullock, William R., M.D., Wilmington, Del.

1887. Bunting, Ross R., M.D., Physician to St. Timothy's Hospital, Roxborough.

1870. Burnett, Charles H., M.D., Professor (Emeritus) of Otology in the Philadelphia Polyclinic; Clinical Professor of Otology in the Woman's Medical College; Aural Surgeon to the Presbyterian Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb, West Philadelphia Hospital for Women, Pennsylvania Epileptic Hospital and Colony Farm and the Bryn Mawr Hospital.

1892. Burr, Charles W., M.D., Professor of Mental and Nervous Diseases in the Medico-Chirurgical College; Neurologist to the Philadelphia Hospital.

1886. Cadwalader, Charles E., M.D.

1895. Carpenter, John T., M.D.

*1897. Carter, William S., M.D., Professor of Physiology in the University of Texas.

1892. Cattell, Henry W., A.M., M.D., Director of the Josephine M. Ayer Clinical Laboratory of the Pennsylvania Hospital; Pathologist to the Philadelphia Hospital.

*1892. Cerna, David, M.D., Ph.D., Galveston, Texas; Demonstrator of Physiology in the Department of Medicine of the University of Texas; Corresponding Fellow of the Sociedad Española de Higiene of Madrid.

1885. Chapin, John B., M.D., Physician-in-Chief to the Pennsylvania Hospital for the Insane.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College.

1897. Chestnut, J. H. W., M.D., Visiting Physician to the Penn Widows' Asylum.

1868. Cheston, D. Murray, M.D.

1897. Cheston, Radcliffe, M.D.

1899. Clark, John G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital.

ELECTED

1873. Clark, Leonardo S., M.D.

1897. Claxton, Charles, A.M., M.D.

1872. Cleemann, Richard A., M.D.

1896. Cleveland, Arthur H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Surgeon-in-Charge of the Ear, Nose and Throat Dispensary of the Presbyterian Hospital.

*1842. Clymer, Meredith, M.D., New York.

1871. Cohen, J. Solis, M.D., Professor (Emeritus) of Diseases of the Throat and Chest in the Philadelphia Polyclinic; Professor (Honorary) of Laryngology in the Jefferson Medical College; Physician to the Home for Consumptives.

1888. Cohen, Solomon Solis, M.D., Professor of Medicine and Therapeutics in the Philadelphia Polyclinic; Lecturer on Clinical Medicine in the Jefferson Medical College; Physician to the Philadelphia, the Rush and the Polyclinic Hospitals; Consulting Physician to the Jewish Hospital; Consulting Laryngologist to the Pennsylvania Training School for Feeble-minded Children.

1898. Coles, Stricker, M.D., Demonstrator of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Maternity.

1895. Cross, William A., M.D., Consulting Physician to the Jewish Hospital.

1884. Curtin, Roland G., M.D., Consulting Physician to the Rush, St. Timothy's and Douglass Hospitals.

1884. Da Costa, John C., M.D., Gynecologist to the Jefferson Medical College Hospital; Consulting Gynecologist to St. Agnes's Hospital.

1896. Da Costa, John Chalmers, M.D., Clinical Professor of Surgery in the Jefferson Medical College; Surgeon to the Philadelphia and St. Joseph's Hospitals.

†1858. Da Costa, J. M., M.D., LL.D., Professor Emeritus; Physician to the Pennsylvania Hospital; Consulting Physician to the Children's Hospital, etc.

1887. Daland, Judson, M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; Professor of

ELECTED

Clinical Medicine in the Philadelphia Polyclinic; Consulting Physician to the Kensington Hospital for Women.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1896. Davis, Charles N., M.D., Assistant Physician to the Dispensary for Diseases of the Skin, and Assistant Surgeon in the Dispensary for Genito-Urinary Diseases in the Hospital of the University of Pennsylvania; Physician to the Department for Skin Diseases of the Northern Dispensary.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College; Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Attending Obstetrician to the Jefferson, Philadelphia and Polyclinic Hospitals.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Goettingen), M.R.C.S. Eng., Assistant Professor of Applied Anatomy in the University of Pennsylvania; Surgeon to the Episcopal, St. Joseph's and Orthopædic Hospitals.

1894. Deaver, Harry C., M.D., Surgeon to the Episcopal, St. Mary's and Samaritan Hospitals and to St. Christopher's Hospital for Children.

1887. Deaver, John B., M.D., Surgeon-in-Chief to the German Hospital; Consulting Surgeon to the Germantown Hospital.

1892. Deaver, Richard Wilmot, M.D.

1885. Dercum, Francis X., M.D., Clinical Professor of Neurology in the Jefferson Medical College; Neurologist to the Philadelphia Hospital; Consulting Neurologist to St. Agnes's and the Jewish Hospitals, and to the State Asylum for the Chronic Insane of Pennsylvania.

1891. Dixon, Samuel G., M.D., President and Executive Curator of the Academy of Natural Sciences of Philadelphia; Member of the Council of the American Philosophical Society; Member of the Board of Managers of the Ludwick Institute, Wistar Institute of Anatomy, the Grandow Institute, Board of Education of Philadelphia, and of the Directors of the Zoölogical Society of Philadelphia; Vice-President of the Pennsylvania Antituberculosis Society.

1891. Dixon, William C., M.D., Physician to the Industrial Home for Blind Women; Physician to the Shelter for Colored

ELECTED

Orphans; Member of Consulting Staff of the Philadelphia Home for Incurables; Examiner of Insane Patients to the Philadelphia Hospital.

1896. Donnellan, P. S., M.D., L.R.C.S. and P., Ireland, Laryngologist to St. Agnes's Hospital; Medical Examiner of the Mutual Life Insurance Company of New York.

1897. Dorland, W. A. Newman, M.D., Instructor in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Demonstrator of Obstetrics in the University of Pennsylvania.

1893. Downs, Norton, M.D.

1864. Downs, R. N., M.D.

1884. Drysdale, T. M., M.D.

1864, Duer, Edward L., A.M., M.D., Gynecologist to the Presbyterian Hospital; Consulting Obstetrician to the Maternity Hospital and to the Preston Retreat.

1897. Duer, S. Naudain, M.D., Physician to the Dispensary of the Presbyterian Hospital.

1871. Duhring, L. A., M.D., Professor of Skin Diseases in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Lecturer on the History of Medicine in the University of Pennsylvania; Surgeon to the Rush Hospital.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D., Rosny sous Bois (Seine), France.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

1899. Edsall, David L., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Associate of the Pepper Laboratory of Clinical Medicine; Physician to the Home for Incurables and to St. Christopher's Hospital for Children; Pathologist to the Methodist Hospital.

*1887. Edwards, William A., M.D., Physician to the Coronado Hospital, Coronado, California.

1896. Ely, Thomas C., A.M., M.D.

1893. Eshner, Augustus A., M.D., Professor of Clinical Medicine

ELECTED.

in the Philadelphia Polyclinic; Physician to the Philadelphia Hospital.

*1880. Eskridge, J. T., M.D., Neurologist to St. Luke's Hospital, and Consulting Alienist and Neurologist to the Arapahoe County Hospital, Denver, Colorado.

1868. Evans, Horace Y., M.D., Physician to the Charity Hospital.

1894. Faries, Randolph, M.D., Surgeon to the Orthopedic Dispensary of the Hospital of the University of Pennsylvania; Director of Physical Education in the Protestant Episcopal Academy.

1893. Farr, William W., M.D., Assistant Physician in the Dispensary for Diseases of the Nose and Throat in the Hospital of the University of Pennsylvania.

1884. Fenton, Thomas H., M.D., Medical Director and Senior Ophthalmologist of the Union Mission Hospital; Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home and to the House of the Good Shepherd.

1884. Fisher, Henry M., M.D., Physician to the Episcopal Hospital; Physician to the Out-patient Department of the Pennsylvania Hospital.

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in the Jefferson Medical College; Clinical Surgeon to the Jefferson Medical College Hospital.

†1885. Fox, Joseph M., M.D., Leesburg, Va.

1897. Frazier, Charles H., M.D., Surgeon to the Philadelphia and Howard Hospitals; Assistant Surgeon to the Hospital of the University of Pennsylvania; Surgeon to the Home for Crippled Children; Assistant Instructor in Clinical Surgery in the University of Pennsylvania.

†1890. Freeman, Walter J., M.D, Professor of Laryngology in the Philadelphia Polyclinic; Laryngologist to the Children's and Orthopædic Hospitals; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1893. Friebis, George, M.D., Ophthalmic Surgeon to the Lutheran Home and Orphanage, Mt. Airy.

1899. Furness, William H., 3d, M.D.

ELECTED

1889. Fussell, M. Howard, M.D., Chief Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania; Instructor in Clinical Medicine in the University of Pennsylvania.

1899. Gamble, Robert G., M.D., One of the Attending Physicians to the Bryn Mawr Hospital.

1873. Gerhard, George S., M.D.

1864. Getchell, F. H., M.D.

1892. Gibb, Joseph S., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose and Throat Department of the Episcopal Hospital.

1899. Gibbon, John H., M.D., Assistant Surgeon to the Jefferson Medical College Hospital; Surgeon to the Out-patient Departments of the Pennsylvania, Children's and Jefferson Hospitals; Surgical Registrar to the Philadelphia Hospital.

1897. Girvin, John H., M.D., Instructor in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Surgeon to the Dispensaries for Diseases of Women of the University Hospital and of the Presbyterian Hospital.

1885. Girvin, Robert M., M.D., Gynecologist to the Presbyterian Hospital.

1889. Githens, William H. H., M.D., Visiting Physician to the Sheltering Arms.

1894. Gleason, E. B., M.D., Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon-in-Charge of the Nose, Throat and Ear Department of the Northern Dispensary.

*1893. Gobrecht, William H., M.D., Washington, D. C.

1884. Godey, Harry, M.D.

1893. Goodell, W. Constantine, M.D.

†1897. Gould, George M., A.M., M.D.

1894. Graham, Edwin E., M.D., Clinical Professor of Diseases of Children in the Jefferson Medical College; Physician to the Franklin Reformatory Home.

1885. Graham, John, M.D.

1891. Green, Walter D., A.M., M.D.

ELECTED

1870. Grier, M. J., M.D.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the University of Pennsylvania; Physician to St. Agnes's, the Children's and the Methodist Hospitals.

1871. Grove, John H., M.D., Consulting Surgeon to St. Agnes's Hospital.

*1889. Guitéras, John, M.D.

*1893. Hamill, Robert H., M.D., Summit, N. J.

1894. Hamill, Samuel McC., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania; Physician to St. Christopher's Hospital for Children; Pediatrist to the Howard Hospital; Member of the American Pediatric Society.

*1859. Hammond, William A., M.D., Surgeon-General U. S. Army (retired), Washington, D. C.

1897. Hand, Alfred, Jr., M.D., Physician to the Out-patient Department of the Methodist, Children's and Polyclinic Hospitals; Pathologist to the Children's Hospital.

1886. Hansell, Howard F., M.D., Clinical Professor of Ophthalmology in the Jefferson Medical College; Professor of Diseases of the Eye in the Philadelphia Polyclinic; Consulting Ophthalmologist to the Chester County Hospital and to the Frederick Douglass Memorial Hospital.

1889. Hare, Hobart A., M.D., Professor of Therapeutics in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Eye Hospital and to the Eye and Ear Department of the Pennsylvania Hospital; Professor (Emeritus) of Diseases of the Eye in the Philadelphia Polyclinic; Consulting Ophthalmologist to the Pennsylvania Institution for the Blind and to the Pennsylvania Institution for the Deaf and Dumb.

1885. Harte, Richard H., M.D., Surgeon to the Pennsylvania and Episcopal Hospitals; Consulting Surgeon to St. Mary's and St. Timothy's Hospitals; Demonstrator of Osteology in the University of Pennsylvania.

ELECTED

1888. Hartzell, Milton B., M.D., Instructor in Dermatology in the University of Pennsylvania; Dermatologist to the Methodist Episcopal Hospital.

1872. Hays, I. Minis, M.D.

1882. Hearn, W. Joseph, M.D., Clinical Professor of Surgery in the Jefferson Medical College; Surgeon to the Philadelphia Hospital.

1884. Henry, Frederick P., M.D., Physician to the Philadelphia Hospital; Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1891. Hewson, Addinell, A.M., M.D., Demonstrator of Anatomy in the Jefferson Medical College; Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to St. Timothy's Hospital.

1872. Hinkle, A. G. B., M.D.

1897. Hinkle, William M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory.

1892. Hinsdale, Guy, M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Presbyterian Hospital.

1888. Hirsh, A. Bern., M.D., Physician to the Home for Aged Couples.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia and Howard Hospitals.

1894. Hoch, William R., M.D., Instructor in Laryngology in the University of Pennsylvania; Laryngologist to the Methodist Episcopal Hospital.

1885. Holland, James W., M.D., Professor of Medical Chemistry and Toxicology in the Jefferson Medical College.

†1879. Hopkins, William Barton, M.D., Surgeon to the Pennsylvania Hospital.

1888. Horwitz, Orville, M.D., Professor of Genito-urinary Diseases in the Jefferson Medical College; Surgeon to the Philadelphia Hospital and to the State Hospital for the Insane; Consulting Surgeon to the Hayes Mechanics' Home.

1868. Howell, Samuel B., M.D., Professor of Chemistry in the Medico-Chirurgical College.

1892. Hughes, William E., M.D., Professor of Clinical Medicine

ELECTED

in the Medico-Chirurgical College; Visiting Physician to the Philadelphia and Medico-Chirurgical Hospitals; Pathologist to the Presbyterian Hospital.

1898. Hutchinson, J. P., M.D., Assistant Demonstrator of Surgery in the University of Pennsylvania; Surgeon to the Dispensaries of the Episcopal, Methodist and Children's Hospitals.

1871. Ingham, James V., M.D.

*1885. Jackson, Edward, M.D., Denver, Colorado, Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic.

1887. Jayne, Horace, M.D., Ph.D., Professor of Zoölogy in the University of Pennsylvania; Director of the Wistar Institute of Anatomy and Biology.

1898. Johnson, Russell H., M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb.

1899. Jopson, John H., M.D., Assistant Demonstrator of Surgery in the University of Pennsylvania; Surgeon to the Dispensaries of the Episcopal, Presbyterian and Children's Hospitals; Visiting Physician to the Philadelphia Home for Incurables.

1885. Judd, Leonardo Da Vinci, M.D.

1886. Jurist, Louis, M.D.

†1867. Keen, William W., M.D., LL.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital; Consulting Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, to St. Agnes's Hospital and to the Woman's Hospital; Membre Correspondant Étranger de la Société de Chirurgie de Paris; Membre Honoraire de la Société Belge de Chirurgie.

1897. Kelly, Aloysius O. J., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Clinical Professor of Pathology in the Woman's Medical College of Pennsylvania; Visiting Physician to St. Mary's and St. Agnes's Hospitals; Pathologist to the German Hospital.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.

ELECTED

1898. Kempton, Augustus F., M.D.

1844. King, Charles R., M.D., Andalusia, Pa.

1895. Kneass, Samuel S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania.

1897. Kyle, D. Braden, M.D., Clinical Professor of Laryngology, Rhinology and Otology in the Jefferson Medical College; Consulting Laryngologist, Rhinologist and Otologist to St. Agnes's Hospital and to the Philadelphia Home for Incurables; Laryngologist to the New Jersey Training School for Feeble-minded Children; Bacteriologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

*1892. Lainé, Damaso T., M.D., Havana, Cuba.

1865. La Roche, C. Percy, M.D.

1887. Leaman, Henry, M.D.

1893. Le Conte, Robert G., M.D., Surgeon to the Methodist Episcopal Hospital and to the Out-patient Departments of the Pennsylvania and Children's Hospitals; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Gynecean Hospital.

1883. Leffmann, Henry, M.D.

1892. Leidy, Joseph, M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital.

1886. Lloyd, J. Hendrie, M.D., Neurologist to the Philadelphia Hospital; Physician to the Methodist Episcopal Hospital and to the Home for Crippled Children; Consulting Neurologist to the State Asylum for the Chronic Insane of Pennsylvania and to the Pennsylvania Training School for Feeble-minded Children.

1893. Longaker, Daniel, M.D.

1877. Longstreth, Morris, M.D.

1886. MacCoy, Alexander W., M.D., Surgeon for Diseases of the Nose and Throat in the Out-patient Department of the

ELECTED

Pennsylvania Hospital; Member of the American Laryngological Society.

1896. Makuen, G. Hudson, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Laryngologist to St. Mary's Hospital and to the Frederick Douglass Memorial Hospital; Visiting Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children.

*1885. Mallet, John William, Ph.D.(Goett.), M.D., LL.D., F.C.S., F.R.S., Member of the Chemical Societies of Paris, Berlin and New York, etc.

1898. Marshall, George Morley, M.D., Laryngologist to the Philadelphia Hospital; Physician and Laryngologist to St. Joseph's Hospital.

1893. Marshall, John, M.D., Professor of Chemistry and Toxicology in the University of Pennsylvania.

1889. Martin, Edward, M.D., Surgeon to the Howard, St. Agnes's, Philadelphia and Bryn Mawr Hospitals; Clinical Professor of Genito-Urinary Diseases in the University of Pennsylvania.

1885. Mays, Thomas J., M.D., Professor of Diseases of the Chest and of Experimental Therapeutics in the Philadelphia Polyclinic; Visiting Physician to the Rush Hospital.

1875. McClellan, George, M.D., Consulting Surgeon to the Howard Hospital.

1895. McFarland, Joseph, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College.

1868. Mears, J. Ewing, M.D..

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D., Columbia, Pa.

1894. Miller, D. J. Milton, M.D., Physician to the Episcopal Hospital; Assistant Physician to the Children's Hospital.

1881. Mills, Charles K., M.D., Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania; Clinical Professor of Neurology in the Woman's Medical College of Pennsylvania; Neurologist to the Philadelphia Hospital.

†1888. Mitchell, John K., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assist-

ELECTED

ant Neurologist to the Presbyterian Hospital; Attending Physician to the Pennsylvania Training School for Feeble-Minded Children.

1856. Mitchell, S. Weir, M.D., LL.D. (Cambridge, Harvard and Princeton); M.D. *Honoris Causa* (Bologna, Italy); Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Presbyterian Hospital.

1882. Montgomery, Edward E., M.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson and St. Joseph's Hospitals.

1863. Morehouse, George Read, M.D., Ph.D. (Princeton), Consulting Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1886. Morris, Caspar, M.D.

1893. Morris, Elliston J., M.D., Physician to the Episcopal Hospital, the Sheltering Arms and the Midnight Mission.

1883. Morris, Henry, M.D., Visiting Physician to St. Joseph's Hospital.

1856. Morris, J. Cheston, M.D.

1897. Morton, Samuel W., M.D.

1861. Morton, Thomas G., M.D., Senior Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to the Jewish Hospital; Emeritus Surgeon to Wills Eye Hospital; Consulting Surgeon to the State Hospital for the Chronic Insane at Wernersville, Pa., to the West Philadelphia Hospital for Women, and to the Pennsylvania Epileptic Hospital and Colony Farm; Honorary Member Société de Médecine Mentale, Belgium; Corresponding Member of the British Orthopædic Society, London.

1891. Morton, Thomas S. K., M.D., Professor of the Principles and Practice of Surgery and of Clinical Surgery in the Woman's Medical College of Pennsylvania; Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Consulting Surgeon to the Philadelphia Dispensary and to the House of Refuge; Surgeon to the Polyclinic Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases.

ELECTED

1864. Moss, William, M.D.

1898. Muehleck, George A., M.D., Pathologist to St. Agnes's Hospital and Chief of Medical Dispensary of the same.

1890. Müller, Auguste F., M.D., Attending Physician to the Germantown Hospital.

1882. Musser, John H., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Philadelphia and Presbyterian Hospitals; Consulting Physician to the Woman's Hospital and the West Philadelphia Hospital for Women.

1896. Myers, T. D., M.D.

1886. Neff, Joseph S., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-Urinary Surgery in the Philadelphia Polyclinic; Assistant Demonstrator of Surgery in the University of Pennsylvania.

1899. Nicholson, William R., Jr., M.D., Instructor in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Obstetrician to the Maternity Hospital; Assistant Surgeon to the Gynecean Hospital.

1889. Noble, Charles P., M.D., Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary; Surgeon-in-Charge of the Department for Women of the Union Mission Hospital; Clinical Professor of Gynecology in the Woman's Medical College of Pennsylvania; Lecturer on Gynecology in the Philadelphia Polyclinic.

1893. Noble, William H., M.D.

1898. Nolan, Edward J., M.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia.

1869. Norris, Herbert, M.D.

1865. Norris, Isaac, M.D.

1892. Norris, Richard C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician-in-Charge to the Preston Retreat; Visiting Obstetrician to the Philadelphia Hospital; Gynecologist to the Methodist Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital.

ELECTED

1866. Norris, William F., M.D., Professor of Ophthalmology and Clinical Professor of Diseases of the Eye in the University of Pennsylvania; Surgeon to Wills Eye Hospital.

1884. Oliver, Charles A., M.D., Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia and the Presbyterian Hospitals; Consulting Ophthalmic Surgeon to St. Agnes's, St. Timothy's and Maternity Hospitals; Consulting Ophthalmologist to the State Hospital for the Chronic Insane of Pennsylvania.

1884. O'Neill, J. W., M.D.

*1885. Osler, William, M.D., Professor of Medicine in Johns Hopkins University and Physician to the Johns Hopkins Hospital, Baltimore, Md.

1897. Packard, Francis R., M.D., Dean of the Philadelphia Polyclinic and College for Graduates in Medicine; Instructor in Laryngology in the University of Pennsylvania; Assistant Physician in the Dispensary for Diseases of the Nose and Throat in the Hospital of the University of Pennsylvania; Surgeon for Diseases of the Ear in the Out-patient Department of the Pennsylvania Hospital; Laryngologist and Otologist to the Pennsylvania Institution for the Deaf and Dumb.

1890. Packard, Frederick A., M.D., Visiting Physician to the Pennsylvania, Philadelphia and Children's Hospitals.

1858. Packard, John H., M.D., Late Surgeon to the Pennsylvania Hospital; Surgeon Emeritus to St. Joseph's Hospital.

1898. Page, Henry F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Instructor in Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to the Baptist Home.

1882. Parish, William H., M.D., Professor of Obstetrics in the Dartmouth Medical College; Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Obstetrician to the Lying-in Charity Hospital; Consulting Surgeon to the Kensington Hospital; Medical Director and Gynecologist to St. Agnes's Hospital.

1899. Parke, William E., M.D., Assistant Surgeon in the Department for Diseases of Women of the Northern Dispensary;

ELECTED

Clinical Assistant and Surgeon to the Dispensary of the Kensington Hospital for Women.

1898. Pearce, F. Savary, M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Chief of the Medical Dispensary of St. Agnes's Hospital.

†1889. Penrose, Charles Bingham, M.D.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania.

1884. Perkins, Francis M., M.D., Ophthalmic Surgeon to St. Agnes's Hospital.

1899. Phillips, John L., M.D.

1890. Phillips, J. Willoughby, M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital; Consulting Physician to the Philadelphia Dispensary and to the Educational Home for Boys.

1896. Posey, Wm. Campbell, M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to the Howard and Epileptic Hospitals and the Home for Incurables; Consulting Ophthalmologist to the State Hospital for the Insane at Norristown.

1885. Potter, Thomas C., M.D.

1899. Potts, Charles S., M.D., Instructor in Nervous Diseases in the University of Pennsylvania; Physician to the Dispensary for Nervous Diseases and Assistant Neurologist to the University Hospital; Consulting Physician to the Hospital for the Insane of Atlantic County, New Jersey; Consulting Neurologist to the State Penitentiary, Eastern District of Pennsylvania.

†1899. Price, Joseph, M.D., Obstetric Physician to the Philadelphia Dispensary.

1887. Randall, B. Alexander, M.D., Clinical Professor of Diseases of the Ear in the University of Pennsylvania; Professor of Diseases of the Ear in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's and Methodist Hospitals; Otologist to the Rush Hospital.

ELECTED

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1897. Rhein, John H. W., M.D., Medical Electrician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children; Instructor in Neuro-pathology in the Philadelphia Polyclinic and College for Graduates in Medicine; Chief of Clinic for Nervous Diseases of St. Agnes's Hospital.

1891. Rhoads, Edward G., M.D.

1898. Riesman, David, M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Demonstrator of Pathological Histology in the University of Pennsylvania; Consulting Physician to the Jewish Hospital; Neurologist to the Northern Dispensary; Visiting Physician to the Northern Day Nursery.

1895. Ring, G. Oram, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic and Aural Surgeon to the Samaritan Hospital.

1891. Risley, S. D., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Attending Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Consulting Ophthalmologist to the New Jersey Training School for Feeble-minded Children; Member of the Board of Managers of the Pennsylvania Training School for Feeble-minded Children.

†1878. Roberts, John B., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic; Surgeon to the Methodist Hospital.

1899. Roberts, Walter, M.D., Instructor in Otology in the Philadelphia Polyclinic; Physician to the Ear, Nose and Throat Department of the Dispensary of St. Christopher's Hospital for Children; Clinical Assistant for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital.

*1888. Robins, Robert P., M.D.

1898. Ross, George G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same.

ELECTED

1897. Sailer, Joseph, M.D., Associate in the Pepper Clinical Laboratory in the University of Pennsylvania; Pathologist to the Pennsylvania Training School for Feeble-minded Children.

†1866. Schäffer, Charles, M.D.

1899. Schamberg, Jay F., M.D., Associate in Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Union Mission Hospital.

1887. De Schweinitz, George E., M.D., Professor of Ophthalmology in the Jefferson Medical College; Consulting Ophthalmic Surgeon to the Philadelphia Polyclinic; Ophthalmic Surgeon to the Philadelphia Hospital; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Ophthalmologist to the Bryn Mawr Hospital and to the Chester County Hospital.

1895. Scott, J. Alison, M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Visiting Physician to the Pennsylvania Hospital; Physician to the Church Home for Children.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb.

1888. Seltzer, Charles M., M.D.

1875. Seyfert, Theodore H., M.D.

1884. Shaffner, Charles, M.D., Ophthalmic Surgeon to the Presbyterian Hospital.

1887. Shakespeare, Edward O., A.M., M.D., Ph.D.

1897. Sharpless, W. T., M.D., Physician to the Chester County Hospital, West Chester, Pa.

1876. Shippen, Edward, A.M., M.D., Medical Director U. S. Navy (retired).

1891. Shober, John B., M.D., Gynecologist to the Philadelphia and Howard Hospitals; Assistant Gynecologist to the Gynecean Hospital.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Methodist Hospital.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms and to the Southern Home for Destitute Children; Assistant Physician to the German Hospital; Physi-

ELECTED

cian to the Out-patient Departments of the German and Pennsylvania Hospitals.

†1896. Shoemaker, William T., M.D., Assistant Ophthalmologist to the German Hospital and Ophthalmic Surgeon to the Out-patient Department of the same; Ophthalmic Surgeon to the Out-patient Department of the Presbyterian Hospital; Ophthalmologist to the Southern Home for Destitute Children.

1880. Simes, J. H. C., M.D., Emeritus Professor of Genito-Urinary and Venereal Diseases in the Philadelphia Polyclinic.

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the State Asylum for the Chronic Insane of Pennsylvania.

1895. Slocum, Harris A., M.D., Professor of Gynecology in the Philadelphia Polyclinic; Gynecologist to St. Clement's Hospital for Epileptics.

*1863. Smith, A. K., M.D., U. S. A. (retired), New York.

*1864. Smith, Edward A., M.D., New York.

1895. Spellissy, Joseph M., M.D., Visiting Surgeon to St. Mary's Hospital; Surgeon to the Out-patient Departments of the Pennsylvania, Methodist and St. Agnes's Hospitals; Assistant Surgeon to the Orthopedic Dispensary of the University Hospital.

1897. Spiller, William G., M.D., Professor of Diseases of the Nervous System in the Philadelphia Polyclinic and College for Graduates in Medicine; Associate in the William Pepper Clinical Laboratory of the University of Pennsylvania; Neurologist to the New Jersey Training School for Feeble-minded Children; Pathologist to the Pennsylvania Training School for Feeble-minded Children; Pathologist to the Pennsylvania Epileptic Hospital and Colony Farm.

1894. Stahl, B. Franklin, Ph.G., B.S., M.D., Instructor in Physical Diagnosis, and Lecturer on Dietetics of the Sick in the University of Pennsylvania; Visiting Physician to St. Agnes's Hospital; Neurological Registrar to the Philadelphia Hospital.

1875. Starr, Louis, M.D.

1898. Steele, J. Dutton, M.D., Instructor in Medicine, and Students' Physician in the University of Pennsylvania; Bac-

ELECTED

teriologist to the Presbyterian Hospital; Physician to the Church Home for Children.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia and the Jewish Hospitals.

1884. Stelwagon, Henry W., M.D., Ph.D., Clinical Professor of Dermatology in the Jefferson Medical College; Dermatologist to the Philadelphia Hospital; Physician to the Department for Skin Diseases of the Howard Hospital; Clinical Professor of Dermatology in the Woman's Medical College; Consulting Dermatologist to the Pennsylvania Institution for the Deaf and Dumb; Socio Corrispondente di La Societa Italiano di Dermatologia e Sifilografia.

1895. Stengel, Alfred, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, Philadelphia and Children's Hospitals.

1888. Stewart, David D., M.D., Professor in the Philadelphia Polyclinic; Attending Physician to the Episcopal Hospital; Consulting Physician to the Kensington Hospital for Women.

1898. Stiles, George M., M.D.

†1842. Stillé, Alfred, M.D., LL.D., Professor (Emeritus) of the Theory and Practice of Medicine in the University of Pennsylvania; Consulting Physician to the Maternity Hospital, to the Woman's Hospital, and to the Rush Hospital.

1898. Stout, George C., M.D., Surgeon-in Charge of the Throat, Nose, and Ear Department of St. Mary's Hospital; Laryngologist and Aurist to the Children's Aid Society; Instructor in Otology in the Philadelphia Polyclinic.

1884. Stryker, Samuel S., M.D., Physician to the Presbyterian Hospital.

1898. Sweet, William M., M.D., Associate in Ophthalmology in the Philadelphia Polyclinic; Instructor in Ophthalmology, and Chief Clinical Assistant in the Out-patient Eye Department of the Jefferson Medical College; Ophthalmic Surgeon to the Phœnixville Hospital.

1886. Taylor, John Madison, M.D., Neurologist to the Howard Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Physician to

ELECTED

the Children's Hospital; Professor of Children's Diseases in the Philadelphia Polyclinic.

1887. Taylor, William J., M.D., Surgeon to St. Agnes's Hospital and to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1886. Taylor, William L., M.D.

1867. Thomas, Charles Hermon, M.D.

1897. Thomson, A. G., M.D., Ophthalmic Surgeon to the Children's Hospital; Assistant Ophthalmic Surgeon to Wills Eye Hospital; Assistant Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases.

†1869. Thomson, William, M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College; Emeritus Surgeon to Wills Eye Hospital.

1896. Thorington, James, A.M., M.D., Adjunct Professor of Diseases of the Eye in the Philadelphia Polyclinic; Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to the Methodist Orphanage; Ophthalmologist to the Pennsylvania Training School for Feeble-minded Children and to the New Jersey State Institution for Feeble-minded Children.

1898. Thornton, Edward Q., M.D., Demonstrator of Therapeutics in the Jefferson Medical College.

1896. Toulmin, Harry, M.D., Assistant Medical Director of the Penn Mutual Life Insurance Company.

†1894. Tunis, Joseph Price, M.D., Formerly Assistant Demonstrator of Anatomy and of Surgery in the University of Pennsylvania; Surgeon to the Methodist Hospital.

1866. Tyson, James, M.D., Professor of Medicine in the University of Pennsylvania; Physician to the Hospital of the University of Pennsylvania and to the Philadelphia Hospital; Consulting Physician to St. Mary's Hospital and to the Kensington Hospital for Women.

1897. Tyson, T. Mellor, M.D., Assistant Physician to the Hospital of the University of Pennsylvania; Physician to the Rush Hospital, the Philadelphia Lying-in Charity Hospital and the Children's Aid Society of Philadelphia.

1864. Vandyke, Edward B., A.M., M.D.

1873. Van Harlingen, Arthur, M.D., Emeritus Professor of Dis-

ELECTED

eases of the Skin in the Philadelphia Polyclinic; Consulting Dermatologist to the Children's Hospital.

1893. Vansant, Eugene Larue, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose and Ear Department of the Howard Hospital.

1897. Veasey, Clarence A, M.D., Adjunct Professor of Diseases of the Eye in the Philadelphia Polyclinic and College for Graduates in Medicine; Demonstrator of Ophthalmology in the Jefferson Medical College; Chief Clinical Assistant to the Ophthalmological Department of the Jefferson Medical College Hospital; Consulting Ophthalmologist to the Philadelphia Lying-in Charity Hospital.

†1883. Vinton, Charles Harrod, M.D.

1885. Walker, James B., M.D., Ph.D, Consulting Physician to the West Philadelphia Hospital for Women and Children.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Laryngologist to the Howard Hospital and to the Hospital for Diseases of the Lungs, Chestnut Hill.

1886. Watson, Edward W., M.D.

1875. Webb, William H., M.D.

1883. Welch, William M., M.D., Physician-in-Charge of the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and to the Northern Home for Friendless Children.

1897. Wells, William H., M.D., Adjunct Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Demonstrator of Clinical Obstetrics in the Jefferson Medical College.

1893. Westcott, Thompson S., M.D., Instructor in Diseases of Children in the University of Pennsylvania; Visiting Physician to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital.

1884. Wharton, Henry R., M.D., Demonstrator of Surgery in the University of Pennsylvania; Surgeon to the Children's and

ELECTED

Presbyterian Hospitals; Consulting Surgeon to the Bryn Mawr Hospital.

1878. White, J. William, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital.

1898. Whiting, Albert D., M.D., Assistant Surgeon and Registrar to the German Hospital, and Surgeon to the Out-patient Department of the same; Physician to the Southern Home for Destitute Children.

†1880. Willard, De Forest, M.D., Clinical Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the White and the Colored Cripples' Homes and to the Home for Incurables.

*1878. Williamson, Jesse, M.D., Wilmington, Delaware, One of the Surgeons to the Delaware Hospital.

1881. Wilson, H. Augustus, M.D., Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Jefferson Medical College; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital and to the Kensington Hospital for Women.

1874. Wilson, James C., M.D., Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician-in-Chief to the German Hospital; Attending Physician to the Pennsylvania Hospital.

1897. Wilson, W. Reynolds, M.D., Visiting Physician to the Philadelphia Lying-in Charity Hospital.

†1884. Wirgman, Charles, M.D., Physician to the Jefferson Medical College Hospital and to the Howard Hospital; Physician to the Out-patient Department of the Children's Hospital.

1893. Wolff, Lawrence, M.D., Formerly Demonstrator of Chemistry in the Jefferson Medical College, Visiting Physician to the German Hospital and Clinical Professor of Medicine in the Woman's Medical College.

1893. Wood, Alfred C., M.D., Instructor in Clinical Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to the Philadelphia Hospital.

1865. Wood, Horatio C., M.D., LL.D. (Yale and Lafayette); Pro-

ELECTED

fessor of Materia Medica, Pharmacy and General Therapeutics in the University of Pennsylvania, and Clinical Professor of Diseases of the Nervous System in the Hospital of the same; Member of the National Academy of Science.

1880. Woodbury, Frank, M.D., Associate in Laryngology in the Philadelphia Polyclinic.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1888. Woodward, Charles E., M.D., Secretary of the West Chester Board of Health; U. S. Examining Surgeon; Member of the Medical Staff of the Chester County Hospital.

†1897. Woodward, George, M.D.

1860. Wurts, Charles Stewart, M.D.

1868. Yarrow, Thomas J., M.D.

1889. Young, James K., M.D., Instructor in Orthopedic Surgery in the University of Pennsylvania; Assistant Orthopedic Surgeon to the University Hospital; Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania.

1894. Zentmayer, William, M.D., Assistant Surgeon to Wills Eye Hospital; Ophthalmologist to St. Mary's Hospital and to the House of Refuge.

1899. Ziegler, S. Lewis, M.D., Ophthalmic Surgeon to St. Joseph's Hospital.

1887. Ziegler, Walter M. L., M.D.

1895. Zimmerman, Mason W., M.D., Ophthalmic Surgeon to the Germantown Hospital and to St. Christopher's Hospital for Children.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

American.

ELECTED

1876. Billings, John S., M.D., U. S. A. (retired), New York.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1896. Conner, Phineas Sanborn, M.D., Cincinnati, Ohio.
1893. Councilman, William T., M.D., Boston, Massachusetts.
1876. Davis, N. S., M.D., Chicago, Illinois.
1886. Draper, William H., M.D., New York.
1892. Emmet, Thomas Addis, M.D., New York.
1892. Fitz, Reginald H., M.D., Boston, Massachusetts.
1895. Fletcher, Robert, M.D., Washington, D. C.
1891. Jacobi, A., M.D., New York.
1893 Kerr, John G., M.D., Canton, China
1895. McBurney, Charles, M.D., New York.
1886. McGuire, Hunter, M.D., Richmond, Virginia.
1876. Moore, E. M., M.D., Rochester, New York.
1886. Reeve, John C., M.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Chicago, Illinois.
1896. Sternberg, George M., M.D., U. S. A., Washington, D. C.
1886. Thomas, T. Gaillard, M.D., New York.
1896. Tiffany, L. McLane, M.D., Baltimore, Maryland.
1894. Warren, J. Collins, M.D., Boston, Massachusetts.
1894. Weir, Robert F., M.D., New York.
1892. Welch, William H., M.D., Baltimore, Maryland.
1886. Whittaker, James T., M.D., Cincinnati, Ohio.

Foreign.

ELECTED

1873. Acland, Henry W., M.D., F.R.S., Oxford, England.
1890. Baccelli, Guido, Rome, Italy.
1877. Barnes, Robert, M.D., London, England.
1894. Brunton, Sir T. Lauder, M.D., London, England.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.
1899. Fraser, Thomas R., M.D., LL.D., F.R.C.P., F.R.S., Edinburgh, Scotland.
1883. Heath, Christopher, F.R.C.S., London, England.
1896. Jaccoud, Prof. S., M.D., Paris, France.
1874. Jackson, J. Hughlings, M.D., London, England.
1893. v. Jaksch, Rudolf, M.D., Prague, Bohemia.
1896. Leyden, Ernst, M.D., Berlin, Germany.
1877. Lord Lister, M.D., LL.D., F.R.S., London, England.
1873. Ogle, John W., M.D., London, England.
1898. Roddick, Thomas G., M.D., Montreal, Canada.
1896. Pye-Smith, P. H., M.D., London, England.
1896. Stewart, Sir T. Grainger, Bart., M.D., Edinburgh, Scotland.
1869. Valcourt, Th. de, M.D., Cannes, France.
1892. Virchow, Rudolf, M.D., Berlin, Germany.

CORRESPONDING MEMBERS.

ELECTED

1880. Carrow, Flemming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Lall, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.

NECROLOGICAL LIST.

FELLOWS.

JUSTUS DUNOTT,	November, 1889
A. C. DEAKYNE,	February 2, 1899
ROBERT P. HARRIS,	February 20, 1899
JOSEPH J. KIRKBRIDE,	May 4, 1899
ROBERT B. CRUICE,	September 14, 1899
ALBERT FRICKE,	November 17, 1899
EMIL FISCHER,	December 12, 1899

ASSOCIATE FELLOWS.

A. M. POLLOCK,	June 20, 1892
SIR GEORGE JOHNSON,	June 3, 1896
SIR JAMES PAGET,	December 30, 1899

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MEMOIR OF LAWRENCE SAVERY SMITH, M.D.

By CHARLES H. FRAZIER, M.D.

[Read January 28, 1899.]

One of the most lamentable and heart-rending features of our recent international war was the havoc played by disease among our troops. How many a young soldier, inspired with a sense of true patriotism, willing and anxious to fight for his country's honor, was stricken down with disease and died ere one opportunity arose to test his valor. Pitiful, too, the fate of those who were not called upon to face the dangers of the firing-line, yet did their country faithful service in fighting the fevers that wrought this havoc among our soldiers, and finally themselves fell victims to disease.

Such was the fate of our late Fellow, Dr. Lawrence Savery Smith. His career in the United States Volunteer Army during the war with Spain was brief. In the early part of May, 1898, at Mount Gretna, Pa., he was mustered in with his command, the First Regiment of the National Guard of Pennsylvania, whose surgeon he had been for several years. When called upon to offer his services to the United States Government and to his regiment, his sense of responsibility and duty prompted him to accept without hesitation the call of his country. Yet at that time it was not a necessary or an easy step to take. It was generally accepted that there was little probability of the volunteer regiments seeing active service; besides, there were many medical men, with little or no responsibilities at home, who were anxious to secure appointments; and Dr. Smith was at this time laying a founda-

tion for a good practice and felt that, if he went off then, he would lose all the ground he had gained, and have to begin afresh on his return. There were other reasons, too, of a personal nature, which made it particularly hard at this juncture of his life to voluntarily leave his home and enter into a life of hazards. While these circumstances weighed heavily on his mind, they never once made him falter when he was called upon to swear allegiance to his country.

On May 13th, his regiment, now known as the First Pennsylvania Volunteers, proceeded under orders to Camp Thomas, Chickamauga, a camp which afterward proved to be one of the most unhealthy of those established during the war. In his care of the sick Dr. Smith was greatly hampered by the woful lack of medical supplies, and appealed repeatedly and in vain to his superior officer. On June 6th Major Smith was detached from his regiment and appointed Surgeon-in-Charge of the Reserve Hospital, First Army Corps. He at once engaged himself busily in organizing his new command and in making it as efficient as possible, before it would be called into active service. It was not long before the call came, for on July 23d orders were received to break camp and proceed to Newport News, where, five days later, he embarked with his command on the transport-ship "Massachusetts," bound for Porto Rico. At this very time, the fever, which was soon to take his life, was already well advanced. The onset of his illness dates back to July 19th, when, on returning from a drill, he had a severe chill. From this time on to the day of embarkation he was anything but well, but, busy with the responsibilities of his new duties and unwilling to give up just when his services would most be needed, he gave little thought to his own condition, and sailed away on the overcrowded transport. Had he been a private in the ranks he would never have been allowed to proceed on the voyage, but would have been ordered to the hospital, for he was already in the second week of typhoid fever. His condition gradually grew worse, and on the arrival of the transport-ship at Ponce, Porto Rico, August 4th, he was removed in an ambulance to a dwelling-

house, where Dr. Joseph P. Tunis, then in charge of the Reserve Ambulance Corps, administered faithfully to his wants. For various reasons it was considered advisable to transfer the patient from these quarters to the hospital-ship "Relief," then lying at anchor in the harbor, and, on August 10th, General Brooke gave orders to that effect. There is little left to tell: four days later the "Relief" sailed away, bound for New York, and when it had proceeded but twenty-four hours, Dr. Smith died, Sunday, August 15, 1898. Such was his brief but useful career in the United States Volunteer Army

Lawrence Savery Smith, the second son of Dr. Albert H. Smith, also a Fellow of the College of Physicians, was born in Philadelphia, April 3, 1868. Descended from Quaker ancestors, who were among the early settlers of Pennsylvania, he represented the third generation of physicians in his family. His grandfather, Dr. Moses B. Smith, graduated from the University of Pennsylvania in 1804, and his father, the late Dr. Albert H. Smith, received his diploma from the same school in 1856.

Lawrence received his early schooling at the Cheltenham Military Academy, where he was prepared for entrance to the College Department of the University of Pennsylvania, and, receiving his degree of B. A. in 1888, he entered the Medical Department in the fall of the same year, and graduated in 1891. After serving as Resident Physician in both the University and Pennsylvania Hospitals, he went abroad in the fall of 1893 to pursue a course of study in pathology and gynecology. At Göttingen he received instruction from Orth in pathology, and in the spring directed his attention chiefly to gynecology, at the University of Berlin. During this time he studied the German language assiduously, and became sufficiently familiar with it to enable him afterward to read German literature with ease. He frequently took advantage of his knowledge of the language, and found therein a source of much pleasure, as well as profit.

In the fall of 1894 he returned to Philadelphia, opened an office, and began the practice of medicine, purposing to make

a specialty of gynecology and obstetrics. It was not long before he was appointed Chief of the Gynecological Dispensary of the University Hospital and Assistant Clinical Instructor of Gynecology in the Medical Department of the University of Pennsylvania, positions which he filled with credit both to himself and to the institutions which he served. On October 14, 1896, he was unanimously elected by the Board to fill a vacancy on the staff of the Maternity Hospital where his services were highly appreciated. Though his connection with this institution was brief, the Board passed resolutions "expressing their sincere sorrow at the loss sustained, not only by the hospital, but by the medical profession and the community, in the death of Dr. Smith, a man of great ability in his profession, a patriot whose death was directly caused by disease contracted in the service of his country in the war with Spain." During the few brief years of his professional career he devoted much of his spare time, that which was not taken up in actual practice, to studying the literature of the branches in which he was interested, and in this way made himself thoroughly conversant with, and kept himself well posted on, every phase of the subject.

Such, briefly told, is a sketch of the professional career of one who was stricken down at the very outset of a career which was destined to make its influence felt for the good of the community in which he lived. I can but regard it as a privilege to have been called upon by your President to write the memoir of our late Fellow, and yet, though perhaps qualified to do so, by virtue of our intimate associations during his professional career, and a friendship begun in his early college days, I feel that I can but feebly portray his character, possessed of so many worthy traits. Above all, he was in his dealings with his patients, as well as his fellow men, honest in the strictest sense of the word. Always having the courage of his convictions, he never hesitated to give expression to them, wholly regardless of the consequences to his personal welfare. This trait of his character, no doubt, oftentimes worked to his material disadvantage. However, his disgust

for those who sought by intrigue to gain advancement or seek recognition in the eyes of the world was such that he never resorted to measures, that might be called politic, to further his own interests, and I know of more occasions than one in which, for just such reasons, he lost opportunities which would have resulted in his own advancement. His absolute regard for the truth and his frankness were conspicuous in his everyday life, and though these attributes are perhaps not calculated to earn for a man what the world calls popularity, yet they are ornaments of a character that cannot but be respected, and in due course of time would receive the recognition they deserve.

From the blood of two generations of physicians he seemed to inherit many of the qualifications of a successful practitioner. The responsibility that every physician must assume by virtue of his profession was never lost sight of, and whether in attendance on the rich or poor, and of the latter he visited not a few, he gave to both the best of his services. Paying strict attention to detail, and conscientious in its execution, he aimed to leave undone nothing, that the patient's means or the circumstances would allow, which would add to his or her comfort and welfare. His natural feeling of sympathy, his frankness, the deep interest he took in each case, and his high sense of responsibility rendered him especially fit for the practice of his profession and won for him a tender regard on the part of his patients. A man of rather domestic tendencies, his circle of acquaintances was not large, but those who knew him well, as I did, found in him a sterling friend and a pleasant companion.

In the opening exercises of the Medical Department in October, 1898, the Provost of the University, referring to those who had died during the war in the service of the country, spoke of Dr. Smith as a "man of high personal character and professional ability," and, emphasizing the earnestness with which he spoke, said in conclusion, "these are not perfunctory words of speaking nothing but good of those who are deceased, but they are words of truth and soberness." So, too, what I

have written in picturing to you his character has been written in all soberness, feeling that in the loss of Dr. Smith we have lost a Fellow whose influence has and would have helped to preserve that high standard of moral responsibility which alone can claim for our profession the respect of the layman, and who, had he lived, would in the future have brought naught but credit to the profession in which he was enrolled as a member. In conclusion, I cannot refrain from quoting the words of others who wrote of him thus: "Honest and strong in manhood, noble in character, benevolent and tolerant in disposition, his death has removed from our profession a spirit which in its every effort exemplified the true ideal of the physician. He pursued his professional work and scientific studies with consistent and progressive efforts. He will live long in the memory of those who knew him."

MEMOIR OF ISAAC MASSEY, M.D.

By WILLIAM T. SHARPLESS, M.D.

[Read April 5, 1899.]

Dr. Isaac Massey was born in Chester County, Pennsylvania, near the borough of West Chester, February 15, 1836. His ancestors were for the most part Quaker farmers of English descent, though his great-grandfather, Jacob Vogdes, probably of Mennonite ancestry, served in Colonel William Montgomery's Chester County battalion of the Flying Camp in the Revolutionary War.

These ancestors were men of simple tastes, industrious habits, and of sturdy independence and uprightness of character.

Dr. Massey grew up on his father's farm, a shy, gentle, studious boy, loving study rather than farm work, and giving up the latter when about twenty years of age to teach a public school in the neighborhood. His previous education had been received in the district schools and at boarding school at Ercildoun and Norristown. He was especially proficient in mathematics, and a little later he was engaged to teach this branch at Wyer's West Chester Academy, where he remained for several years.

At one time he had made arrangements for entering Yale College in order to prepare himself better for the profession of teaching, but this was given up for the study of medicine, and he entered the Jefferson Medical College in 1862.

During the summer of 1863, though he had had but one year of study, he served as Assistant Surgeon of the Twenty-

ninth Regiment of the Pennsylvania Volunteer Militia, and was mustered out with his regiment when the emergency which culminated in the battle of Gettysburg was passed.

He graduated with the first honors of his class in 1864, and re-entered the army as Assistant Surgeon, being stationed on the Pacific coast. A year later he left the army, and in the autumn of 1865 he settled in the practice of medicine in West Chester. He was particularly well qualified for general practice, and the profession he had chosen was exactly calculated to develop what was best in him. His kindly, modest bearing, and his unassumed personal interest in his patients at once gained their confidence, overcame their natural reserve and placed them at their ease. He was always dignified and well bred, and this dignity and breeding came from no careful practice of a studied form, but it was the natural result of a disposition thoughtful for the interests and feelings of others and of a heart overflowing with kindness for everyone.

His unselfish devotion to his patients was very remarkable. No one was too poor, no service too menial, no fatigue too great. The simple fact that some one was sick and asked for his aid placed upon him a solemn obligation. No one recognized more fully than he the claim that the poor have upon the profession, and no one has been more faithful to this trust. Among his patients were many people of good family, whose income had been reduced or their support removed, but who were still anxious to pay him for his attendance. He managed to give gratuitous service to these with a tact so delicate that the recipients of it were scarcely able to realize that he was conferring a favor.

He had unusual clearness of perception, and his mental processes were quick and accurate. As he lived in a small town, where specialists are not within easy reach, his labors covered almost every field of medical work, with equal success in all, and his therapeutic resources seemed almost inexhaustible.

Perhaps there is no occupation so well calculated to develop absolute self-reliance as the practice of medicine in the coun-

try. The most serious emergencies must be met promptly, and they must be met alone. For this sort of practice sound judgment and available knowledge, based upon experience, are of more value than the most complete familiarity with the details of modern laboratory work. The former qualities Dr. Massey possessed, and he did not undervalue the latter.

Although he was a director in several business and charitable enterprises, and was School Director of West Chester for nearly twenty years, he had few interests outside of his practice, which became in time one of the largest and most lucrative in the county.

He was a member of the Chester County and Pennsylvania State Medical Societies and of the American Medical Association. In 1887 he was elected a Fellow of this College. For thirty-one years he was the physician to the Friends' Boarding School at Westtown. At the time of his death he was consulting physician to the Oakbourne Epileptic Colony and to the House of Refuge at Glen Mills. In 1892 he was elected a manager of the latter institution to fill the vacancy caused by the death of D. Hayes Agnew. In this institution he felt the greatest interest, serving on important committees of the Board and giving to it freely of his time.

In 1893, when the Chester County Hospital was organized, he was elected one of the original medical staff, and he continued to serve the institution faithfully until his death.

In person Dr. Massey was singularly attractive. His tall, spare figure and vigorous constitution were capable of great physical exertion with little apparent effort. I believe it is safe to say that in the thirty-four years of his practice he did not average more than six hours' sleep per day. He enjoyed almost uninterrupted good health until his death, which occurred from apoplexy, February 18, 1898, at the house of a patient where he was making a professional call. He survived the original attack but a few hours, and died in his sixty-third year. A widow and one daughter survive him.

Dr. Massey was not an original investigator. He was not a medical author or teacher. He was a practitioner of medicine.

His influence and reputation were limited to those with whom he came into personal relation. He was one of that class from whom, on account of close daily contact, the public forms its estimate of the medical profession. That profession has been dignified in our community by his private life and professional career.

BOWEL-RESECTION; END-TO-END SUTURE. KRASKE'S OPERATION; END-TO-SUTURE OF RECTUM. PYLORIC DILATATION WITH SUBSEQUENT GASTROENTEROSTOMY AND PYLOROPLASTY.

By JOHN B. DEAVER, M.D.,
CHIEF SURGEON TO THE GERMAN HOSPITAL.

[Read January 4, 1899.]

I HAVE thought the following cases of sufficient interest to record, particularly as they demonstrate that mechanical appliances are not absolutely necessary to obtain good results in the respective operations. I believe that the simpler the technique of any operation the nearer it approaches the ideal. It will be noted in the histories of the cases that bowel-resection, with end-to-end approximation, was practised in three cases, end-to-end approximation in one case of ruptured bowel, and lateral anastomosis in one; in all of these recovery, with restoration of function, followed. In the case of transverse tear of the first portion of the rectum in the operation for pyosalpinx, only the Trendelenburg position rendered effective suturing of the bowel-rent possible.

A. D., a white female, fifty-eight years old, was admitted to the German Hospital on May 20, 1896, with strangulated femoral hernia. Operation revealed gangrenous bowel, six inches of which were resected, and end-to-end anastomosis effected. Recovery uninterrupted.

R. J., a white female, fifty-three years old, was admitted to the German Hospital on March 14, 1898. Two years previously she had been operated upon for an umbilical hernia of nineteen years' standing, and she had since had trouble in the movement of her bowels. Two weeks before admission she was suddenly seized with violent abdominal pain and vomiting. The

vomiting continued at intervals of about three or four hours. After ten days the vomit was of a fecal character. The bowels were moved by an enema on the ninth day, and wind was passed by the bowel quite easily during the whole of her illness. On admission the patient was given an enema, which was followed by a free, well-formed movement.

Under ether an incision six inches in length was made in the median line of the abdomen, excising the scar left from the former operation. A stricture of the small bowel, not entirely occluding the lumen, was found. Four inches of bowel were resected and end-to-end anastomsis was effected. The abdomen was closed with wormgut. Recovery followed.

C. W., a white male, fifteen years old, was admitted to the German Hospital on April 6, 1898, with the following history: While riding a bicycle around a street-corner, the street being wet, he was thrown upon the street-car track, along which was passing a large four-wheel wagon, pulled by two horses, one of the front wheels of the wagon passing over his abdomen. Upon admission to the hospital the boy was found to be in great pain, with some shock. Examination of the belly-walls yielded negative information other than the presence of pronounced rigidity. I was in the hospital at the time the boy was admitted, and it was my judgment, from the presence of excessive pain and the great degree of rigidity of the belly muscles, that the patient was suffering from a serious intra-abdominal lesion. I advised immediate operation, which was agreed to. There was found a complete transverse tear of the small bowel, the ends of which were trimmed with scissors and united end to end. With the exception of an attack of regurgitant vomiting, which occurred on the second day, recovery was uninterrupted.

Mr. L., a white male, forty-five years old, was admitted to the German Hospital in April, 1898, with a diagnosis of carcinoma of the upper and of the terminal portion of the rectum, for the relief of which a modified Kraske operation was undertaken, with excision of the involved bowel, cutting wide of the diseased area, and end-to-end anastomosis. The wound was packed with gauze, and healing was complete. The result was perfect, with restoration of normal bowel-movement through the anus.

L. E., a white female, twenty-four years old, was admitted to the German Hospital on July 23, 1897, with general peritonitis and the added diagnosis of double pyosalpinx. She had been married two years, and had had no children and no miscarriage, never having been pregnant. Menstruation was irregular and painful. A vaginal discharge had begun after marriage, with soreness and inflammation of the genitals and burning on micturition. The woman had had severe colicky pains deep in the pelvis and over the lower part of the abdomen. The stooping position caused discomfort, and there was general abdominal tenderness. On vaginal examination, a mass was found behind and on either side of the uterus, high up.

Abdominal section was performed and the uterine appendages removed.

Dense adhesions were found involving the upper portion of the rectum. In the enucleation of the inflammatory appendages the rectum was torn across, and the rent was repaired. Glass drainage was employed, and recovery was uneventful.

A. —., a white male, twenty-two years old, while at sea was the subject of an irreducible hernia, which subsequently became strangulated. Upon reaching port, five days later, he was operated upon for strangulated hernia. The bowel was found to be doubtful in appearance; yet the operator returned it to the abdomen and closed the incision. The patient complained of great pain for several days, when the wound opened and the entire bowel involved in the former hernia was discharged as a slough. A large fecal fistula resulted, for which I subsequently effected intestinal anastomosis, with the introduction of the segmented rubber rings. The condition of the bowel at the site of the fistula, which involved the small bowel, was as follows: The bowel to the proximal side of the fistula was greatly distended while the part beyond was exceedingly contracted: it was with great difficulty that the anastomosing ring could be introduced into the latter.

While the operation of the anastomosis was in part successful, a fecal fistula persisted, and as but one of the segmented rings was discharged per rectum, a second operation was performed for the double purpose of closing the remaining small fecal fistula and removing the unexpelled segmented ring. This operation resulted in complete cure. It is now three years since the operation.

M. C., a white female, aged twenty-eight years, was admitted to the German Hospital on June 13, 1898, with a diagnosis of pylorie stricture. The patient had swallowed a partial set of artificial teeth three years previously, which was subsequently passed by the bowel. Three months later she complained of pain in the stomach, which increased paroxysmally at intervals for fifteen months, since which time the pain had been more or less constant. Vomiting set in one year after the teeth had been swallowed; and since then the patient had been able to retain only liquid food. Blood was found in the vomitus. A localized point of tenderness was detected over the upper portion of the right rectus muscle. Abdominal section, with gastrotomy, was performed on June 30, and a pyloric stricture of small calibre was corrected by pyloroplasty. Recovery was uneventful.

T. G., a white male, forty-eight years old, was admitted to the German Hospital on June 4, 1898, with a diagnosis of non-malignant pyloric obstruction. Abdominal section, with gastrotomy, was performed, and dilatation of a pylorie stricture practised. Prompt recovery followed. On October 17, 1898, gastro-enterostomy was performed by direct apposition, using needle and thread only. Death occurred on the fourth day from rupture of an abscess of the lung. Autopsy showed the absence of peritonitis, with water-tight communication between the stomach and the small

bowel. The right lung was the site of an abscess-cavity. The left lung was diseased at its apex.

It is interesting to note that the pyloric orifice was found as it was left at the previous operation, patulous to the normal degree. This case will be reported in detail later.

The results obtained in the foregoing cases, as reported in this communication, and in other cases, from direct approximation after bowel-excision, as well as in lateral anastomosis, without the use of mechanical appliances, the facility and the rapidity with which this can be done without instruments other than needle and thread, warrant a declaration in favor of this simple method of technique. The time consumed in completing an anastomosis by this method is from ten to fifteen minutes under favorable circumstances.

Prior to the case reported of lateral anastomosis with segmented rubber rings it had been my practice to employ mechanical devices. After the unpleasant experience with this case I decided to discard mechanical means and simply use needle and thread. The results obtained have been so satisfactory as to justify me in advocating this method as superior to others.

The method of end-to-end approximation with sutures is likewise employed by me in my operations for lateral anastomosis.

After a limited experience with the operation of dilatation for the relief of non-malignant pyloric obstruction I have decided that pyloroplasty promises a more permanent result.

The question of preliminary colotomy for carcinoma of the rectum, when Kraske's operation is indicated, is, I believe, a debatable one, particularly in the class of cases in which the growth can be removed without sacrificing the terminal portion of the rectum, including the anus.

From the stand-point of safety against peritoneal infection I have not found it necessary to perform preliminary colotomy. It is my practice to first explore the seat of disease, by removal of part of the sacrum, including the coccyx, etc., when it can be determined if the amount of involvement will necessitate the sacrifice of so much bowel as to preclude the possibility of uniting the divided ends. In the presence of disease too extensive to warrant an attempt at excision and end-to-end suture, anterior (iliac) colotomy

is immediately performed. The colotomy having been completed, the patient's general condition influences me either to excise the diseased rectum now or to defer this for a few days. When the affected bowel is excised the peritoneum is opened and the upper portion of the rectum, with the mesorectum and enough of the mesosigmoid, is drawn down to allow of easy apposition of the ends of the divided rectum. The parietal peritoneum is stitched to the mesosigmoid and peritoneal covering of the sigmoid; this shuts off the peritoneal cavity. It is my practice to reinforce the line of suture with a temporary packing of gauze, when I am able to complete the operation—excision, and suture or closure of the upper end of the bowel by invagination—with but little risk of infecting the peritoneum.

DISCUSSION.

Dr. W. J. Taylor: The surgeon can accomplish, in my opinion, the very best results with the needle without any form of mechanical appliance. I have had so little personal experience in using mechanical devices that I hesitate to say anything about that proposed by Dr. Deaver. I am, however, open to conviction that it is as efficient as the device or appliance of Dr. Laplace, in which I have been very much interested.

EXPERIENCES IN THE HOSPITALS OF PHILADELPHIA WITH TYPHOID FEVER ORIGINATING AMONG THE SOLDIERS IN THE LATE WAR WITH SPAIN.

[Read February 1, 1899.]

At the University of Pennsylvania.

By JAMES TYSON, M.D.

During the late summer and autumn of 1898 there were admitted to the Hospital of the University of Pennsylvania 112 cases of typhoid fever from the various military camps of the country. A few of the cases admitted as typhoid fever are omitted from this report because of the absence of distinctive symptoms or because convalescence was so far established that they illustrated no symptoms and required no treatment.

The average age of 105, whose age was secured, was 23¼ years.

Of the 112 cases, 107 recovered and 5 died, say 4.5 per cent., actually a little less, leaving 95.5 per cent. of recoveries.[1] Of the cases admitted, 77, or 68 per cent., reported headache among the earlier symptoms; 19, or 16 per cent., vertigo; 19, or 6 per cent., nosebleed, and 19, or 16 per cent., nausea and vomiting. In 61, or 54 per cent., there was diarrhœa; 29, or 26 per cent., were constipated in the beginning and more or less throughout, while in 22, or 19 per cent., no allusion is made to these states. There was so much confusion in the reports of chills and chilliness that they cannot be regarded as reliable. Pain in the back and limbs was reported in 57, or 50 per cent. Rose-

[1] The total number of cases in which a primary diagnosis of typhoid fever was made was 145. Some of these proved to be other affections; others, as stated, were so far convalescent from typhoid that it was thought not fair to include them, though had they been included the percentage of mortality would have been less.

colored spots were noted by us in 60, or say, 53.5 per cent., absent in 52, or 46.5 per cent. Hemorrhage of the bowels occurred in 9, or 8 per cent., and perforation followed by peritonitis and death in 1 case. Of the cases of hemorrhage 2 died and 7 recovered, and the perforation alluded to was the cause of death in 1 of these. Tympanitic distention of the abdomen was present in 96 cases, or 85 per cent.; dryness of the tongue and sordes in 6, or 5.3 per cent. Delirium was noted in 18, or 15 per cent. The spleen was enlarged appreciably to percussion or palpation in 62, or 55 per cent.

The Widal reaction was found in 71 cases out of 92 in which it was tried, or 77 per cent.; in 2, or 2.1 per cent., it was doubtful, and in 19, or 20.7 per cent., it failed. In 20 it was apparently not tried. In 10 cases the test was made twice, in 1 three times. Of those tried twice, 7 were positive both times; 2 were negative at first and positive at the second; 1 was positive at first and negative the second. In the one in which it was done three times it was negative at the first and positive at the second and third. The tests were made partly in the City Laboratory and partly in the William Pepper Clinical Laboratory. We are inclined to believe that the number of positive reactions in the latter would have been larger but for the fact that one of the cultures used proved to be defective. The stage of the illness at which the test was made was very different, and in most cases it was impossible to ascertain the day of the disease even approximately. The result of our experience with this test has been to increase our confidence in its reliability. It occasionally fails, it may at times appear late, but if obtained by competent, accurate and conscientious observers and is associated with continued fever, I accept it as sufficient evidence of the presence of typhoid fever. Sources of error due to carelessness or incompetency of the person making it should not be regarded as invalidating the test, and I believe that if these elements of error are eliminated the number of cases in which it fails and the number in which it occurs in other diseases likely to be confounded with typhoid fever will be small.

Abscesses or boils were present in 7 cases, or 6.2 per cent.; bedsores in none—a happy event which may be laid to the credit of

modern nursing. Parotitis, which proceeded to suppuration, was present in one case. This patient died on the twenty-first day, and during the last week had symptoms of localized peritonitis. There were 8 cases of thrombosis, or 7 per cent., an unusually large proportion when it is remembered the text-books put it down at 1 per cent. In 6 the left leg was involved; in 2 no mention is made. Some of these cases were quite mild. Localized peritonitis clinically recognized was present in 2 cases, one being associated with appendicitis, of which there was 1 case.

Pneumonia is reported as a complication in 2 cases, 1 of which was fatal; bronchitis in 29, or 26.7 per cent. Albuminuria was noted in but 3 cases, in one of which—a fatal case—it was copious; in 2 it was small, but in one of these it was accompanied by hyaline casts. In a number of cases the urine was not examined, or if examined the results were not recorded, so that our observations in this direction are defective. The malarial plasmodium was reported present on admission in one case, but was not found at the second examination. In this case there were rose-colored spots, large spleen, and the blood responded to the Widal test. In two cases the malarial organism was found during convalescence associated with other symptoms of malaria. Ulcerative tonsillitis occurred in two cases, orchitis in one case, and otitis in one. The relapse remains the enigma of typhoid fever. In the first place, opinion is by no means unmixed as to what constitutes relapse. It is recognized by all that not every rise of temperature in the course of convalescence constitutes a relapse, even if it be of several days' duration. On the other hand, few relapses repeat all the events which go to constitute the primary illness. Finally, it is likely that for some time longer one observer will call that a relapse which another will regard as a simple rise of temperature due to a temporary cause. In other words, the personal equation will always make itself more or less felt. In recognizing the presence of a relapse we have regarded it necessary: first, that the patient's temperature should have been for a time normal; second, that the recurrent fever should be unaccompanied by an intercurrent complication which could cause the fever; and third, that there should be a return of some one or more of the original symptoms,

such as coated tongue, diarrhœa, tympanitic distention of the abdomen, or other evidence of gastro-intestinal derangement. The duration of the relapse may be variable, from a few days to several weeks to be followed by a normal period and second relapse, possibly by another and even fatal relapse. Similar increased febrile movements occurring during the decline of the disease, but before the temperature has reached the normal line, are probably due to the same cause—that is, reinfection. They are, however, not included among our relapses because of the greater difficulty in separating them from the original disease. For them the usual term recrudescence is appropriate. Thus determined there were twelve relapses among our cases, a percentage of 10.7. In two cases at least there were two relapses. In one a supposed third turned out to be a brewing appendicitis, which at one time threatened the life of the patient, but ultimately passed away, seemingly completely, and the patient was discharged after 118 days in the hospital.

The duration of the disease in most cases was very difficult to determine, in some it was impossible because of the circumstances surrounding the initial symptoms. A similar difficulty consequently attends the determination of the *average* duration, to which, therefore, much importance dare not be attached. As far as could be determined, omitting certain cases in which it was impossible to determine even approximately the onset of the illness, the average duration was forty-one and four-fifths days. The average date of the illness at which the temperature first reached the normal in cases of recovery is of much more practical importance and may be set down as nearly as determinable at eighteen and one-half days. Of the fatal cases it has been mentioned that one died of perforation and succeeding peritonitis, death occurring on the twenty-first day. The remaining deaths may be ascribed to exhaustion after long illness or high temperature. The second died on the tenth day after admission and apparently on the tenth day of the disease, having been the subject of high temperature throughout. The third died on the thirty-first day, also exhausted by high temperature. The fourth died on the forty-third day, apparently of toxic adynamia. The case was a nervous one, in which tremor

and weakness were characteristic from the beginning. The pyrexia was never extreme and death was preceded by obstinate vomiting. The fifth fatal case was admitted convalescent on what appeared to be the thirty-sixth day of the disease; relapse began on the forty-second day, pneumonia of the left lower lobe was recognized on the seventy-second day, and he died on the seventy-seventh day of the disease. No autopsies were secured.

The treatment, outside of the dietetic, may fairly be said to have been the cold tub-bath method of Brand. Not every patient was tubbed, for some did not reach the temperature which called for the tubs, namely, 102.2° F. In the majority of cases, on the other hand, the hydrotherapy, by reason of late admission, was instituted much later than its advocates hold should be done in order to secure its best results. I do not insist that the very favorable results were due solely to the Brand treatment. There were undoubtedly a good many mild cases. On the other hand, quite a number of these were thrown out of consideration altogether. My experience with the Brand treatment in this set of cases has in no way diminished my confidence in it as the best available method for treating typhoid fever, subject to the limitations which good sense and experience may demand.

After all, the burning question with this set of cases is the etiology of the disease, for it cannot be said that the symptomatology, course or termination differ largely from those of any other set. A careful record was kept at the University Hospital of the answers to questions as to the source of water supplied to the soldiers in camp, and they included springs, wells, artesian wells, town-supply, creeks, lakes, reservoirs. No large number partook of water from one source more than another, and while contaminated drinking-water doubtless caused some cases, especially the earlier ones, some other cause must be sought for the majority. It is scarcely profitable to discuss this with the scanty data at hand, but it seems reasonable to suppose that transmission through some other agency than drinking-water was also a factor. The more than usual number of medical officers and nurses attacked points to this, while the insurmountable obstacles in the way of disposal of excreta favored it. So far as I know there was no spread of the disease after the

soldiers entered the hospitals, neither nurses nor physicians having been infected. It may reasonably be inferred that the facilities for thorough disinfection and disposal of alvine dejecta were at least in part responsible for this absence of infection. How far that ubiquitous and faithful though illy appreciated scavenger, the *musca domestica*, is responsible, as has been alleged, for the spread of the disease in camps is undetermined, though I see no reason why such a mode should not be admitted. The influence of predisposing causes must not be overlooked, and it is more than likely that the effects of fatigue, indifferent food, and previous illness were a potent factor in favoring the lodgement and development of the infection.

I desire to express, in conclusion, my acknowledgments to Dr. Alfred Stengel and my son, Dr. T. Mellor Tyson, for valuable assistance in collecting the cases and the various symptoms and pathological states which go to make up the report.

At the German Hospital.

By J. C. WILSON, M.D.

The total number of soldiers treated in the German Hospital was 275. Of these 147 suffered from enteric fever. The first soldiers were admitted August 21, 1898; the last were discharged January 2, 1899. Of the 147 cases of enteric fever 5 died—a mortality of 3.4 per cent. The average age of the soldier-patients was 23.5 years. They were all volunteers—in a certain sense picked men, who upon enlistment a few months previously had fulfilled the requirements of a fairly rigorous medical examination.

As bearing upon the period of greatest prevalence of enteric fever in the camps the following figures are of interest:

24, or 16.3 per cent., were admitted during August;

28, or 19 per cent., were admitted during September;

89, or 60.5 per cent., were admitted during October;

8, or 5.5 per cent., were admitted during November.

By far the greater number of these cases were brought from

Camp Thomas and Camp Meade in two trains organized and equipped by the German Hospital authorities. Some were received by allotment from special trains provided by other hospitals or the city. Some few wandered into the hospital.

95, or 64.6 per cent., were from Camp Meade;

37, or 25.2 per cent., were from Camp Thomas, Chickamauga;

4, or 2.7 per cent., were from Camp Poland, Knoxville;

2, or 1.4 per cent., were from Camp Fernandina;

2, or 1.4 per cent., were from Camp Black;

1, or 0.68 per cent., was from Camp Wickoff;

1, or 0.68 per cent., was from Puerto Rico;

5, or 3.4 per cent., were unclassified.

The average duration of the disease upon admission was about 11.3 days.

The date of the beginning of the disease could only be approximately determined in the greater number of the cases. In this respect the cases resembled those encountered in civil practice. The beginning of the attack was reckoned from the day when the patient, having suffered from prodromic symptoms, became incapable of ordinary duty or was known to have fever. In a very large proportion of the cases the facts were ascertained at the time from the patient himself and confirmed by subsequent inquiry during convalescence. In other instances the information was obtained from comrades. In some of the patients the appearance of the rash coincidently with enlargement of the spleen and the subsequent course of the febrile movement were utilized for the estimation of the day of the attack. Some degree of uncertainty attaches in the great majority of instances to the date at which the prodromic symptoms merge into those of the declared disease.

The average day upon which the temperature became normal in 131 cases as recorded was 22.4 from the beginning of the attack, as nearly as could be ascertained. Eleven patients were admitted after the defervescence; 5 died during the course of the fever. The average duration of the stay in the hospital was 31.2 days.

Hemorrhage occurred in 8 cases, or 5.4 per cent. The amount of blood lost in a single stool varied from 30 c.c. to 800 c.c. In

one case which terminated fatally 2600 c.c. of blood were voided in six discharges from the bowels.

Relapse occurred in 21 cases, 14.2 per cent. A single relapse in 18 cases, 12.2 per cent.; multiple relapse—3 attacks, including the primary disease—in 3 cases, 2 per cent. The average length of the single relapses was 11.6 days; the average length of the relapses in the multiple cases 14.3 days. The average length of the afebrile period between the attacks in the cases of single relapse was 5.9 days; the average length of the same period in the cases of multiple relapse was 9.3 days.

Simple toxic or febrile albuminuria without casts occurred in 84 cases, or 57.1 per cent.

Nephritis as shown by the presence of albumin, free blood, and casts of various kinds, was present in 37 cases, or 25.2 per cent.

The diazo-reaction was studied some time during the course of the disease in 87 cases. The result was positive in 61, or 70.2 per cent.; negative in 26, or 29.8 per cent.

The action of the blood-serum upon cultures of the bacillus typhosus—the Widal test—was investigated in all the cases in duplicate, specimens of blood being sent at the same time to the bacteriological laboratory at the City Hall and to the laboratory of the German Hospital. In some few instances there was want of correspondence in the reports, but in most of these, duplicate specimens sent at a later period gave similar results. In five instances the result from both laboratories was negative. In 4 of these cases, however, there could be no doubt about the clinical diagnosis. In the fifth, a case from Camp Meade, the clinical diagnosis was rendered in the highest degree probable by the general symptom-complex and the course of the attack.

Venous thrombosis occurred in 5 cases, 3.4 per cent. In every instances the veins of the left leg only were involved. This accident occurred invariably after the defervescence and during the early course of convalescence and before the patient had left his bed.

There is a clinical history of directly previous malarial infection in 16 cases, 10.9 per cent. The malarial parasite was found in 35 cases, 23.8 per cent. The form was usually non-pigmented, intracorpuscular bodies and free hyaline bodies, and pigment in the

blood. In several of these cases irregular chills developed during convalescence, recovery taking place under treatment by quinine in appropriate doses.

Pleurisy alone occurred in 2 cases, 1.4 per cent.

Croupous pneumonia occurred in 6 cases, 4.1 per cent. In one instance the pneumonia was followed by an empyema, recovery taking place after drainage.

Acute bronchitis, persisting for some time into convalescence, occurred in 2 cases, 1.4 per cent.

Acute lacunar tonsillitis occurred in 2 cases, 1.4 per cent.

Acute laryngitis with aphonia, but without necrosis of the cartilages, and terminating in recovery, occurred in 1 instance, 0.68 per cent.

Otitis media did not occur in any instance.

Acro-paræsthesia occurred in 4 cases, 2.7 per cent.

Severe secondary syphilis, antedating the attack of enteric fever, was present in 1 case, 0.68 per cent.

An acute gonorrhœa was noted in one case, 0.68 per cent.

Of the 5 fatal cases, one patient, aged twenty-eight years, died upon the twenty-sixth day of the attack and the twentieth day after admission, of croupous pneumonia. A second, aged twenty-one years, died on the twentieth day of the attack, and the ninth day after admission, from intense infecton. A third, aged twenty-one years, died on the twenty-sixth day of the attack, and the nineteenth day after admission, of peritonitis following repeated profuse intestinal hemorrhage. The fourth, aged twenty-two years, died upon the thirty-first day of the attack, and the twentieth day after admission, of peritonitis following repeated hemorrhages, and the fifth, aged twenty years, was admitted moribund on the twenty-first day of the attack, and died upon the third day with symptoms of perforative peritonitis. In none of these cases was a post-mortem examination made.

Of the 147 cases of enteric fever, 121 were treated by systematic cold bathing, the average number of baths administered being 37.8; 26 cases were not systematically bathed for special reasons; the greater number of them were admitted with falling or already normal temperatures; several were admitted with a history of recent

intestinal hemorrhage, and were, therefore, not bathed, and one was admitted moribund with the symptoms of peritonitis.

The foregoing statistics have been carefully compiled from the bedside records by Dr. Henry F. Page, Assistant Physician to the Hospital.

At the Pennsylvania Hospital.

By ARTHUR V. MEIGS, M.D.

The soldiers suffering with typhoid fever who were sent to the Pennsylvania Hospital by the Government during the late war with Spain were in the charge of several physicians. It will, therefore, be impossible for me to do more than to speak in general terms of the whole number of them, confining myself, when I want to illustrate particular points, to the patients who were in my own care. This method of dealing with the subject ought to give a good general idea of the disease as it manifested itself in our hospital, for it is fair to presume, as I had charge of nearly one-fourth of the total number, that my patients presented most of the varieties of the disease and most of the complications that occurred.

The total number of soldiers received into the Pennsylvania Hospital was 326, of whom 21 died, making a percentage of mortality of 6.44. Two of the deaths were from tuberculosis, and all of the others from typhoid fever. Almost all of the men were affected with acute disease, there being very few chronic cases. There were cases of dysentery and malaria, and men suffering from the effects of yellow fever and other acute diseases which they had had in the South.

The total number of cases of typhoid fever was 214, and the number of deaths 19, making a percentage of mortality from this cause of 8.88. This includes all the cases marked upon the records of the hospital as typhoid fever. A few of these were men nearly convalescent, and they remained in the hospital only a few days, but most of them suffered with the greater portions of their attacks after their admission. They were brought in in every possible stage of the disease, but I have thought that in dealing with the subject

numerically, the only thing that could be done was to give the total number of cases received and the total number of deaths, and from this to derive the percentage of mortality. This is all I shall say about the whole number of cases of typhoid fever in the soldiers received in our hospital except that when I come to speak of the treatment, I shall again make use of the larger figures to illustrate one point.

There came under my own care 48 cases of typhoid fever. Some of the men were brought by ship directly from Porto Rico to this city, others came from Montank Point, Long Island, and still others were from Camp Meade, near Harrisburg, and from other camps throughout the country. Among the men brought from Montank Point there were a great many who had served in Cuba. Of the 48 patients who were in my care, 2 died, making a percentage of mortality of 4.17. One of these deaths was of a man, forty years of age, who said he had been for years a hard drinker. While he was in the hospital there were albumin and casts in the urine, and at the autopsy the liver and spleen were found to be enlarged, to have thickened capsules, and to be adherent to surrounding parts. The kidneys were fibroid, and none of the organs were soft, as is usual in typhoid fever. The mesenteric glands were hardly enlarged, and these were only a few shallow ulcers in the lower part of the ileum; there were not in the Peyer's patches, and were irregularly round in shape. The other death was of a man, twenty years old, who died in collapse after being in the hospital nine days, and in whom the autopsy revealed the existence of perforations of the ileum, through which feces had escaped into the abdominal cavity.

Hemorrhage from the bowel occurred in 5 cases in the hospital, and another of the patients had had a hemorrhage before his admission. There were bed-sores in two cases, bed-sore and abscesses of other parts of the surface in 2, and surface-abscesses in 3 cases. The rose-rash was noted as present in 33 cases. There was albumin in the urine in 24 of the patients, and in many of these casts also were found. In one case casts were found, but no albumin. Tibial periostitis occurred in one case. Crural phlebitis was noted twice, but in neither instance was it severe. Otitis

media occurred once. In two cases there was a return of fever after convalescence seemed to have been fully established. As this seemed to be due to malaria, and both of the patients had been in malarial regions, the blood was examined and the plamodium found. These patients soon recovered under the use of quinine. There can be no doubt, and there never has been in my mind, that typhoid fever and malaria frequently exist in the same patient at the same time. The term typho-malarial fever, therefore, is a good one, for it is descriptive of a condition which is not at all uncommon.

My experience with typhoid fever in soldiers has led me to the conviction that it is in no essential particular different from the same disease as we know it in our city hospitals under ordinary circumstances and in private practice. There is, however, a great difference in the physical condition of the patients. Most of our patients with typhoid fever in city hospitals are people from the poorest and most ignorant class, and they are of very various ages, some very young and some forty or even over fifty years of age. They are often newly arrived immigrants, who have been subject to poverty and hardship all their lives, and many of them have pre-existing chronic disease. Consequently, a large proportion of them have the disease severely, and a large percentage must die. The soldiers, on the contrary, were almost all young men who had been selected for military service after a medical examination of greater or less rigidity, to ascertain that they were physically sound. They were, therefore, a picked body of men of the age most likely to be able to successfully resist attacks of acute disease. Typhoid fever was forced upon their naturally healthy bodies by hardship, improper food, overwork in climates to which they were not accustomed, and exposure to various poisonous influences. Under such conditions it might be expected that as soon as the patients were removed from the influences that had given them the disease, and were well cared for, a large proportion of them would recover, and everyone who has given any attention to the subject knows that this was the case.

One phase of the care of the soldiers afforded me a new experience as a hospital physician, and this was one which probably comes

often to military surgeons in war. I entered my ward one morning to find thirty-six patients I had never seen before, almost all of them very sick, and, therefore, in need of immediate attention. These men had been only a few hours in the hospital, and were brought in without any written records to indicate what had been their previous condition; there was not even so much as a scrap of paper giving a diagnosis. Had I been obliged to set to work unaided to bring order out of such chaos, the labor would have been vastly greater than it proved; but the system existing in a well-ordered hospital had been at work before my arrival. The executive department had provided in advance everything needed for the care of the sick men, and it may be well to say that in our hospital there was no overcrowding. The soldiers were placed only in the regular wards. There were fourteen beds in a ward which ordinarily contains twelve, but in every other respect the quarters of the soldiers were the same as those accorded other patients at ordinary times. The resident physician had already been at work, and had in the short time at his disposal acquired a pretty good idea of the condition of the patients, and there were nurses enough to carry out promptly all directions given. Two or three hours of concentrated labor enabled me to make diagnoses that were sufficiently accurate to permit of the treatment being at once organized. What I saw upon this occasion gave me a better comprehension than I had before of the difficulties that must beset a military surgeon in the field after a battle, or when large numbers of sick and injured men are suddenly taken from one place to another, as is often done in war. We should be careful to ascertain the facts before we criticise operations in war, for often what may at first seem to have been the result of neglect will be found to have occurred because military officers were asked to do what was impossible. Nothing but the excellence of the machinery of our hospital organization and the fact that this was not stretched beyond its capacity for immediate expansion enabled the Pennsylvania Hospital to cope successfully with the emergency. Our managers did not at any time receive more soldiers than we were ready for, and in consequence we were able to give them as good care as is given to other patients in ordinary times.

The first of the soldiers who were consigned to our hospital had been brought directly from Porto Rico. Their physical condition was, in one respect, peculiar; they were almost all very anæmic, and almost all of them had extremely dicrotic pulses. This was as marked among those who had other diseases as among those suffering with typhoid fever. The condition is one that I had not previously met with, although I believe this form of anæmia is well known to military surgeons who have seen service among men of the temperate climates on duty in the tropics.

The Widal test for typhoid fever must not be passed by without mention. I have concluded after careful study of the results obtained from examination of the blood as it is done for us by the health bureau of this city, that as a diagnostic aid it is useless. This conclusion has been forced upon me by the fact that the test has often indicated the existence of typhoid fever in patients who manifestly did not have it, and on the other hand, the test has failed to confirm clinical evidence of the presence of typhoid fever.

The Widal test has often reacted positively in cases of tuberculosis.

My discussion of my experience of the typhoid fever of the soldiers would be incomplete if I failed to mention a case in which I fell into the error of mistaking tuberculosis for typhoid fever. A man, thirty-nine years of age, who had served in Porto Rico and had been sick while there for about three weeks, was brought to the hospital with almost every symptom of typhoid fever. He had diarrhœa and was delirious; the tongue was coated, dry, and brown; there was fever, the pulse was dicrotic, and he was emaciated. There were rose-colored spots upon the abdomen which were exactly like the eruption of typhoid fever. He had had a hemorrhage from the bowel before his coming to the hospital and had two more after his arrival. The Widal test gave a positive reaction upon two occasions. This man soon got better. The temperature fell, the delirium passed away, and it seemed as if he was convalescing, but at the end of about nine days he became worse. It was evident that there was more disease of the lungs than was compatible with the existence of uncomplicated typhoid fever, and he emaciated rapidly. I thought pulmonary tuberculosis had come

upon him as he was recovering from typhoid fever. After twenty-three days in the hospital he died. At the autopsy there were found to be extensive tubercular infiltration of the lungs and numerous minute tubercles in the spleen. The mesenteric glands were little if at all enlarged. The Peyer's patches were not ulcerated, although in the lower three feet of the ileum and the colon there were numerous shallow ulcers. In the ileum these ulcers did not extend in the length of the gut, but were of as great or greater extent around it. The lesions which have been described are those of tuberculosis and not of typhoid fever, and when the case is considered in its entirety it must be classed as having been purely tubercular from the beginning. The similarity of the clinical phenomena with those of typhoid fever was complete, and without the light which was thrown upon the case by the post-mortem examination the error of classing it as typhoid fever could not have been avoided. The differential diagnosis of tuberculosis from typhoid fever is occasionally impossible during life.

The treatment I pursued with my patients was as follows: Absolute confinement to bed; six ounces of milk every three hours; the administration of five minims of dilute muriatic acid every three hours, and sponging of the surface with cool water every three hours if the temperature rose as high as 102° F. The sponging was continued during fifteen minutes. This was the routine treatment, but it was varied to suit the needs of individual cases. In severe cases, when it seemed desirable to give medicines every two hours, the nourishment was also given every two hours, but in that case only four ounces of milk were given at a time. Three pints of milk I consider to be the proper total quantity, and this amount need seldom be exceeded. The food and medicine should be given at like intervals, either every two, three, or four hours, as the requirements of the cases seemed to demand, and the medicine should be given a quarter of an hour after the food. This is as nearly together as it is desirable to give them, and gives afterward the longest period of rest that can be obtained. It is very important that the patient should be disturbed as little as possible, and that the administration of food and medicines at very short intervals should be avoided. The medicines I varied as seemed to be

desirable, using turpentine, carbonate of ammonia, digitalis, spirit of chloroform, or other drugs as it appeared likely their effects might be sustaining to the failing powers of the patients. I tried not to give many drugs at the same time, withdrawing one as another had to be given.

Our hospital afforded an opportunity to compare the results obtained by the use of the cold plunge-bath with those obtained from such treatment as I have described. This comparison is interesting and, I hope, may be instructive, although it cannot be expected to yield evidence which will be accepted as conclusive with regard to the merits of the one method of treatment or the other. It will require more than 214 cases of typhoid fever to put this much-vexed question at rest. The physicians of the Pennsylvania Hospital are divided in their opinions in regard to the use of the cold plunge bath. There were certain wards, therefore, in which the bath was regularly used as a routine method of treatment, and others in which it was not. There were admitted to the wards in which the cold plunge was regularly employed, 95 patients with typhoid fever, of whom 11 died, and there were admitted to the wards where bathing was not employed, 119 patients, of whom 8 died. This made a mortality under the bath treatment of 11.58 per cent. and with baths, a mortality of 6.72 per cent.

It so happened that there was a transfer, at the end of four days after 17 patients were admitted, of one ward from a bath advocate, to another of our physicians who does not bathe. These 17 patients had, therefore, baths for four days after their arrival at the hospital, and during the rest of their stay they were treated without baths. All of them recovered. In the figures, as they have been given, these cases were classified with those that were treated with baths, but if it be considered that they belong rather to the number of those who were treated without baths, as they received baths only during four days and the rest of the time were treated by the other method, it makes quite a difference in the figures and in the resulting percentages. It would then be as follows: Treated with baths, 78; deaths, 11; percentage of mortality, 14.10. Treated without baths, 136; deaths, 8; percentage of mortality, 5.88. These percentages speak very eloquently to me, and although the

number of cases is not sufficiently large to prove anything, a consideration of the results does lend strong moral support to those of us who have never been able to think that the cold plunge-bath treatment could accomplish all that its more enthusiastic advocates have believed.

At St. Agnes' Hospital.

By B. Franklin Stahl, B.S., M.D.

The brief time at my disposal makes it impossible to do more than recite the symptoms common to a large proportion of the patients treated and to dwell at greater length upon one or more of the unusual complications.

There were received into the wards of St. Agnes' Hospital during August, September, and October, the period of my service at the hospital, 413 soldiers. Of this number 144 had typhoid fever. The statements made in this paper are based upon the records of these patients. In addition to the 144 mentioned we received 33 patients who were so far convalescent from typhoid fever when they came under my care as to make them unsuited for admission to the class from which the statistics offered have been drawn.

There is an especial interest in the study of these cases, for the reason that they are picked men, men selected for army service, and it may be assumed that they were in perfect health. Furthermore, they represent a class that should stand for high physical development, by reason of the number being largely made up of men from the regular army and those recruited from the National Guard. This would seem to justify us in expecting them to be better able to withstand the fever they developd at a later period. This would be a reasonable assumption, were it not for the fact that camp-life, as we know it to have been, induced indisposition and invalidism, with the result that many of the soldiers were seriously sick before they developed typhoid fever. Added to this, when we consider the inadequate hospital equipment, the deficient dietary, and, worst of all, the absence of trained nurses, we are prepared to realize that the men received for treatment were very ill and unfavorable subjects for a low mortality.

The ages of the patients treated are of interest: Between the ages of seventeen and twenty years there were 11 patients; twenty and twenty-five years, 91; twenty-five and thirty years, 29; thirty and thirty-five years, 6; thirty-five and forty years, 6, and between forty and forty-five years there was 1 patient.

Taking up the consideration of the temperature of these cases, it is to be noted that a large proportion of the patients had their highest temperature when admitted to the hospital. Thus, 45 of the patients, or 32 per cent., did not have a temperature at any time during their stay in the hospital exceeding that noted on admission. But many of them frequently had a temperature equal to that noted on admission. This would seem to raise the question whether this limitation of the febrile process was due to the stage of the disease at which they were received or to the better facilities for caring for their needs.

The exceptional pyrexia shown by these cases makes me wish that I might have an opportunity to show you a number of the charts and have you study these records of a most interesting group of patients. Some idea of the temperature may be had, however, by a recital of the number showing varying degrees of fever.

Thus, there was 1 patient with a temperature of 107°; there were 3 whose temperature on one or more occasions registered 106°, or more; 31, 105°, or more; 53, 104°, or more; 35, 103°, or more; 14, 102°, or more; 7, 101°, or more.

The occurrence of spots was noted in 130 of the cases, being 89 per cent. of all the cases. The atypical manifestation of the rash consisted in its persistence when the patient was free from fever, in several instances, and in other cases, the profusion of the spots upon the chest, coupled with the paucity of them upon the abdomen.

It is probable that a better idea of the condition of these patients can be had by tabulating some of the most important facts in the records of the series reported:

No	Age	Camp.	Temp on adm.	Pulse	Resp.	Highest temp.
1	22	Fernandina	105°	110	30	107.2
2	24	"	101	90	20	101.6
3	29	"	102.8	100	30	102
4	27	"	102	98	24	103.6
5	20	"	102.2	100	26	102.2
6	23	"	102.2	98	26	104
7	23	"	102	100	25	102
8	21	"	104	110	26	104
9	22	"	104	100	26	104
10	22	"	102	90	24	104
11	23	"	104	110	26	104
12	22	"	102	88	24	104.2
13	28	"	101	90	24	101
14	24	"	102	90	26	105
15	22	"	103	100	26	104
16	34	Meade.	101	90	22	101
17	21	"	101	90	24	103.4
18	27	"	104	90	28	104
19	27	"	101	70	20	104
20	21	"	101	100	22	101.4
21	27	"	102.4	90	24	103.4
22	22	"	100.6	80	26	103.4
23	19	"	102.4	80	18	106.4
24	20	"	104.2	80	20	104.8
25	21	"	101	80	22	103
26	19	"	100	80	20	103.6
27	24	"	101	80	24	103.4
28	22	"	102	100	30	103
29	23	"	103.2	110	24	105
30	25	"	104	100	24	104
31	21	"	104	110	38	104.4
32	19	"	104	100	26	104
33	25	"	104	100	29	104
34	17	"	103	100	26	101
35	27	"	103	100	28	101
36	25	"	101.6	90	24	101.5
37	20	"	103	100	28	103
38	21	"	104	110	26	105
39	22	"	100.8	78	26	102
40	26	"	104	100	24	105
41	20	"	102	110	26	105
42	25	"	102	92	22	103.8
43	22	"	102	88	24	103.6
44	20	"	104.2	108	28	104.8
45	35	"	104	106	22	101.4
46	22	"	102.2	104	24	103.4
47	21	"	104.2	114	28	104.2
48	35	"	102	90	26	103
49	27	"	103	90	26	104.8
50	33	Meade and Black.	104	90	26	104.8
51	35	Meade.	101.8	94	24	103
52	21	"	104	100	24	106
53	24	Meade and Ft. Monroe.	103.8	102	24	103.8
54	21	Ft. Monroe, Meade and Black.	100	82	20	103.8
55	34	Black and Meade.	103.6	90	24	103.6
56	19	Atkinson.	104.8	80	24	104.8
57	19	Meade.	100	90	20	103.8
58	26	"	102	85	26	103.8
59	21	"	104.3	100	24	104.8
60	36	"	102	95	24	103.8
61	24	"	104	90	26	104
62	27	"	100.6	86	26	101.8
63	23	"	101.2	80	24	102.8
64	21	"	102	100	20	103
65	21	Meade.	105°	102	23	105°
66	20	"	103.8	100	30	104.8
67	23	"	104.6	100	20	104.6
68	21	"	102	90	20	102
69	25	"	102.4	95	24	104
70	22	Black and Meade.	104.4	115	26	100.4
71	21	Meade.	103.2	94	26	104.8
72	27	"	102	92	24	104
73	22	"	104.2	100	29	104.2
74	21	Meade and Eaton.	104	100	28	105
75	26	Meade.	105	100	33	105.2
76	23	Meade and Eaton.	105.2	105	26	105.4
77	28	Meade and Black.	101	84	20	103.6
78	25	Meade and Black.	105.2	110	36	105.2
79	21	Meade.	104.8	112	26	104.8
80	24	"	105	120	30	105
81	22	"	105	110	30	105.2
82	20	"	105.2	100	20	105.2
83	29	"	105.4	120	32	106
84	24	Meade and Black.	105	102	22	105
85	38	Meade and Black.	103	110	26	104.2
86	21	Meade.	101.2	78	24	103.2
87	20	"	102	80	24	103.6
88	21	Meade and Black.	103.4	100	22	103.4
89	22	Meade and Black.	101	100	20	102
90	22	Porto Rico.	101.6	85	20	102
91	20	" "	101.4	110	20	102
92	19	Meade.	104	100	26	105
93	19	"	102.4	100	26	105.4
94	42	"	99	70	18	101.2
95	21	"	103	100	26	103.8
96	20	"	101	90	24	102
97	21	"	104	100	30	104
98	24	"	104	100	28	105
99	19	"	105	110	30	105
100	40	"	102.4	90	18	104.2
101	23	"	105	110	26	105
102	23	"	105	100	30	105.2
103	21	"	102	100	22	103.6
104	23	"	102	90	22	102
105	24	"	104	110	30	105
106	19	"	101	85	24	101.4
107	25	"	102.2	85	26	103.6
108	27	"	101	60	18	104
109	22	?	102	95	28	103
110	25	Meade.	102.2	110	24	103.8
111	24	"	100.8	95	28	104.4
112	22	"	105	60	28	105
113	23	"	101	90	24	104
114	19	"	102.4	80	22	104.2
115	31	"	102.2	100	28	104
116	24	?	102	70	26	103
117	20	?	105	100	26	105
118	27	Meade.	102	102	20	103.2
119	30	Meade and Black.	104	110	26	104
120	28	Meade.	103.4	90	24	104.8
121	21	"	103	100	20	105.6
122	34	?	104.4	110	24	105.8
123	21	?	104	110	26	104.8

No.	Age	Camp.	Temp on adm.	Pulse	Resp.	Highest temp.
124	23	M'Pherson & Wykoff	104°	90	22	101°
125	24	?	104	100	30	105
126	18	?	102.4	90	25	102.4
127	21	Black.	102.6	104	24	102.6
128	21	Black and Meade.	104	90	24	104
129	18	Meade.	104.6	100	26	104.6
130	22	"	105	110	24	105
131	24	Meade, Alger and Zures?	104	120	26	104.2
132	23	Meade.	104	102	23	104
133	25	Meade.	105.2°	105	26	105.2°
134	20	"	104.4	115	26	104.4
135	21	"	102.2	105	24	103.4
136	21	"	102.4	104	30	104
137	22	"	102.6	100	24	104
138	27	"	102	94	20	102
139	24	"	104.8	104	24	105
140	24	"	102.8	98	22	103.2
141	25	"	105	90	24	105
142	21	"	102	95	24	104
143	23	"	101	90	26	103
144	21	"	104.2	110	26	104.2

No.	Day of disease.	1st day normal.	Days in house.	No. of baths.	Widal reaction.	Result.
1	?	...	13	58	Yes	Died; autopsy.
2	8	12	10	0	"	Recov.
3	?	?	11	0	"	"
4	18	23	11	3	"	"
5	18	23	9	0	"	"
6	12	18	9	4	"	"
7	21	24	4	0	"	"
8	14	26	19	21	No	"
9	9	19	17	7	"	"
10	14	34	39	3	Yes	"
11	10	33	37	27	"	"
12	14	30	60	13	"	"
13	?	5 days after admiss.	13	0	"	"
14	13	29	41	46	"	"
15	14	20	21	5	"	"
16	10	14	8	0	Had typhoid 5 yrs. ago	"
17	9	18	16	1	"	"
18	7	25	43	33	"	"
19	4	16	26	7	"	"
20	7	15	25	0	"	"
21	10	44	51	6	"	"
22	21	35	25	7	"	"
23	21	21	22	5	"	"
24	10	36	42	41	"	"
25	10	22	23	1	"	"
26	11	29	64	9	"	"
27	?	Preced. by rheumatism	64	16	"	"
28	28	48	25	1	"	"
29	9	35	42	63	"	"
30	14	28	35	32	"	"
31	8	23	43	18	"	"
32	28	39	23	19	"	"
33	21	0	18	36	"	Died; autopsy.
34	14	26	24	10	"	Recov.
35	21	31	24	11	"	"
36	10	28	38	43	Rep. lost	"
37	8	22	21	15	Yes	"
38	14	28	42	38	"	"
39	9	19	23	0	Yes	Recov.
40	31	47	35	55	"	"
41	3	...	9	8	No rep.	Died; autopsy.
42	3	21	38	8	"	"
43	4	32	38	3	"	"
44	8	17	21	8	"	"
45	10	28	41	2	Rep. lost	Recov.
46	9	23	26	22	"	"
47	6	25	33	12	Yes	"
48	14	31	38	3	"	"
49	4	27	39	4	2d test	"
50	?	?	39	5	"	"
51	5	25	35	8	"	"
52	18	...	7	13	Yes	Died.
53	11	21	25	2	"	"
54	11	26	29	1	"	"
55	6	27	30	3	No	"
56	15	23	19	1	"	"
57	8	15	23	1	"	"
58	8	34	32	5	Yes	"
59	10	30	49	14	"	"
60	15	18	46	2	"	"
61	7	21	27	3	"	"
62	9	16	27	0	No	"
63	10	17	27	0	Yes	"
64	8	21	31	0	"	"
65	8	20	41	7	"	"
66	8	21	41	28	"	"
67	14	17	22	3	"	"
68	14	20	18	0	"	"
69	14	31	31	9	Rep. lost	Recov.
70	10	24	38	11	Yes	"
71	14	33	41	33	"	"
72	10	29	46	24	"	"
73	8	21	41	16	"	"
74	12	17	47	5	"	"
75	22	32	46	41	"	"
76	9	32	41	14	"	"
77	10	15	40	5	"	"
78	3	18	38	15	"	"
79	4	20	31	3	"	"
80	10	22	31	15	"	"
81	6	19	45	21	"	"
82	3	17	31	21	2d test	"
83	4	32	38	42	Yes	"
84	4	19	38	12	"	"

No.	Day of disease.	1st day normal	Days in house.	No. of baths	Widal reaction.	Result.
85	14	23	24	1	Yes	Recov.
86	14	23	26	1	"	"
87	3	22	38	6	2d test	"
88	5	20	29	2	No	"
89	21	24	29	0	"	"
90	14	21	20	0	Yes	"
91	40	40	16	0	No	"
92	21	41	115	58	Yes	"
93	?	7 days after admis.[1]	112	[2]	Yes, 3d test	"
94	53	53	15	0	"	"
95	10	19	74	13	"	"
96	21	25	12	0	"	"
97	14	30	31	18	"	"
98	7	...	5	34	"	Died.
99	14	40	56	42	"	Recov.
100	31	45	35	8	Rec. lost	"
101	?	4 days after admiss.	9	5	No	"
102	5	23	36	80	Yes	"
103	8	21	23	4	2d test	"
104	12	26	23	0	"	"
105	21	35	26	35	Yes	"
106	10	19	26	0	"	"
107	28	39	37	13	"	"
108	7	14	16	7	"	"
109	7	18	22	0	"	"
110	21	32	26	2	"	"
111	21	38	35	36	Rep. lost	"
112	14	23	45	25	Yes	"
113	21	39	38	50	Yes	Recov.
114	10	34	75	21	"	"
115	16	22	12	2	"	"
116	8	16	19	1	"	"
117	3	32	53	9	2d test	"
118	8	17	55	1	Yes	"
119	12	27	49	7	"	"
120	8	15	34	2	No rec.	"
121	7	45	68	71	Yes	"
122	6	26	71	16	"	"
123	14	22	18	19	No	"
124	14	21	25	3	Yes	"
125	4	16	46	22	"	"
126	7	11	9	1	"	"
127	18	23	15	1	"	"
128	14	17	18	2	"	"
129	8	24	23	1	"	"
130	9	19	43	4	"	"
131	14	28	39	8	"	"
132	14	35	40	5	"	"
133	8	20	40	2	No rec.	"
134	6	12	31	6	Yes	"
135	6	20	39	6	"	"
136	11	17	38	3	"	"
137	10	27	40	2	"	"
138	14	29	27	0	"	"
139	10	27	47	20	No rec.	"
140	15	29	27	1	Yes	"
141	5	20	39	8	"	"
142	7	16	24	4	No rec.	"
143	10	19	39	1	Yes	"
144	10	20	27	6	"	"

THE WIDAL REACTION. The blood of all these patients was submitted to the bacteriological department of the Board of Health, and the results obtained are recorded in the table submitted. In ten cases we received a negative report, and while the effort was made to secure a second report on all cases reported negatively, I find the record of only seven cases that gave the reaction with the second specimen of blood submitted, and one case that did not give the reaction until the third trial. This last case is of interest, because the symptoms and the development of hemorrhage by this patient convince me that he was about the tenth day of the disease when the third specimen showed the reaction. I regret that the records of ten cases have been lost, and thus it is not possible to

[1] After the temperature had been normal eight days the patient was allowed to drive, with other convalescents, in charge of an army surgeon. He returned to the hospital at 11 P.M., and the next day had a temperature of 105°, and a most protracted illness developed.

[2] This man had 18 sponge-baths, notwithstanding the temperature was above the tubbing-point. He was then given 28 tub-baths, and sponging had to be substituted because of the circulatory and nervous condition of the patient; 14 sponge-baths were given, making a total of 32 sponge, and 28 tub-baths given this patient.

state the proportion of cases in which the reaction was present. The figures I am able to show, it seems to me, are sufficient to justify the great faith I have in this test as an evidence of the presence of the disease.

Effort has been made to determine *the day of the disease*, and, counting from this time, to estimate the first day, the patient had a normal temperature. To my mind both of these points are of necessity so inaccurate as to be of no practical value, the reason being the indifferent health of the patient for a varying time prior to the development of the symptoms ushering in the typhoid fever. The figures submitted are based upon the time stated by the patient to be the date of his indisposition.

The symptoms associated with the digestive tract were much less severe than those we were accustomed to see when these patients were treated without baths.

Diarrhœa. Diarrhœa was of comparatively infrequent occurrence. Indeed, a frequent need was a safe laxative. There being less demand for opiates to control the bowels, the secretions were less disturbed, and the tongue so thick and dry that it could not be protruded from the mouth remains only as a memory of the days when the treatment did not include hydrotherapy.

Moderate tympany was present in a large number of the cases, but seldom became a serious complicating factor.

The mortality was 5 cases in 144, being 2.8 per cent.

Treatment. Every patient having a temperature of 102.2° was bathed every third hour. The only exception to this rule was for a period of a few days when we were pressed by the large number of soldiers admitted, when the bathing-point was made 103°. These patients received 1830 tub-baths. The work implied by these figures will be appreciated as exceptional, and would have been almost impossible of accomplishment without a much larger force of assistants than we had, if it had not been for the kindness of Mr. John L. Morris, who designed and gave the hospital an arrangement that so lessens the lifting work incident to giving a bath that one or two persons are able to bathe a patient with the expenditure of a minimum amount of strength in lifting. The photographs show better than I can tell the plan followed. The

carriage now in use at the hospital is an improvement upon the one shown in the picture, in that it is not higher than the level of the bed. The stretcher on it is the one shown in the picture, and the patient is immersed in the bath without being changed from the stretcher. The carrier is on a railroad, placed near the ceiling, as shown in the picture, and the machinery is so adjusted that a child could do the handling of the patient. The tub is stationary, and thus we are able to change the water for each patient, or bathe with constantly changing water.

FIG 1

Shows the carriage on which the patient is brought from the ward to the bath-room. To the side is seen the bath-tub, and above it the stretcher on which he is lowered into the tub. The stretcher is detachable and can be placed on the carriage, so that the patient does not have to make more than one change instead of two, as was the case before the adoption of this plan.

The patient is given whiskey after the bath, and, if it seems indicated, an additional dose is given.

In only two instances was it necessary to decidedly change the rule for bathing. Both were cases that had weak hearts and later developed gangrene. Thus one could only remain in the bath eight instead of fifteen minutes. The other patient had twenty-eight baths, and then became so nervous as the time for them approached, that they had to be replaced by sponging.

FIG. 2.

Shows the railroad, placed near the ceiling, by means of which the patient is carried horizontally from the carriage to a position over the tub. By means of a separate set of pulleys the patient is lowered into the tub.

FIG. 3.

Shows the patient being lowered into the tub. The curve in the stretcher serves as a pillow.

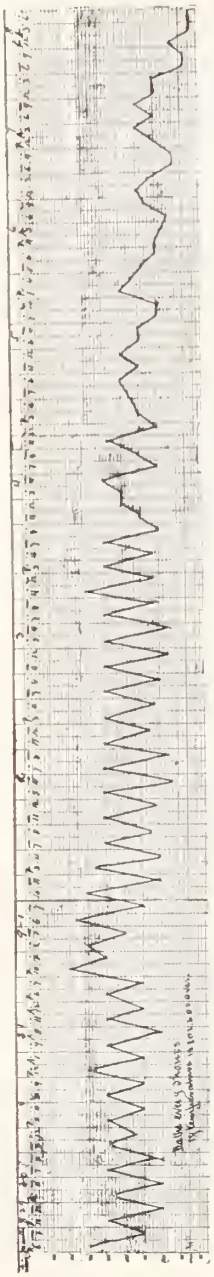

Chart showing a temperature that almost always varied a full degree.

An interesting condition, the explanation of which is not perfectly clear to me, is shown in the chart. I refer to the temperature registering a full degree every third hour. That is, it was never 103° or 104° and a fraction. I show you several of a number of charts in which this peculiarity is exhibited. The most probable explanation that has occurred to me is that the heat production or dissipation was so nearly stable that the same amount was produced in three hours. But this leaves unexplained the difference in the number of full degrees of heat produced.

The medical treatment of the cases consisted in the administration of fractional doses of calomel. At a later period salol with bismuth was given when there was a marked tendency to tympanites. Whiskey and strychnine were given in the later stages, when the condition of the heart indicated the need of them. Turpentine was given when the tongue was dry, thick, and brown, and the change wrought in twenty-four hours was usually so pronounced as to make me marvel that any one treating a large number of typhoid-fever patients should question its value.

Early in my service I lost one of my patients, and the condition of his tissues on section gave me an important therapeutical suggestion. This patient seemed to have almost no liquid element in him. The liver cut like a piece of dried beef, and of similar character were the other organs of the body. This led me to resort to hypodermoclysis and enteroclysis—using for the

purpose a normal salt solution. The results obtained were most gratifying. The mental and circulatory conditions frequently improved with surprising promptness. The patients were encouraged to drink unusually large quantities of water.

Complications. The complications presented by these patients seemed to run the entire gamut, unless we note the absence of bone-involvement. But since this is usually a later complication, it is likely reserved for others to witness the final manifestation of the typhoid fever contracted in the war of 1898.

Blood. The blood of these patients constantly showed a surprising diminution in the hæmoglobin and corpuscular elements.

The changes were out of all proportion to the appearance and symptoms of the patient. Dr. Muerleck, the pathologist to the hospital, made a study of this aspect of these cases and will doubtless make a report of his findings.

Lungs.

Case No.		
1	Pneumonia.	Both lower lobes.
10	"	Catarrhal.
14	"	Left lower lobe, aphonia, hemoptysis.
86	"	Left lower lobe.
38	"	Right lower lobe.
40	"	" " "
41	"	" " "
54	"	" " "
72	"	" " "
75	"	Both upper lobes.
76	"	Right lower lobe.
92	"	Left lower lobe, left upper lobe.
93	"	Left lower lobe.
112	"	" " "
121	"	Catarrhal.
127	"	Left lower lobe.
130	"	" " "

Bronchitis.

Case No.		*Case No.*	
16	Bronchitis.	88	Bronchitis.
24	"	89	"
32	"	95	"
34	"	97	"
35	"	103	"
36	"	79	"
38	"	47	"
39	"	49	"
40	"	56	"

Pleurisy.

74	Pleurisy.	Right.
60	"	Right with effusion.
67	"	" " "

Heart.

Case No.		*Case No.*	
64	Pericarditis.	142	Pericarditis.
74	"	61	"
114	"		

Irregular or Intermittent.

18	Irregular.
119	Intermittent. Second aortic accentuated.
126	" " " " "
114	" " " " "
92	Irregular.
93	"
122	At mitral area First week, second accentuated.
96	" " " " " "
97	" " " " " "
69	" " " " " "
34	" " " " " "
76	" " " " " "
40	" " First week.

Vertigo.

Case No.		*Case No.*	
19	Vertigo.	96	Vertigo.
92	"	100	"

Mitral Systolic Murmur.

36	Mitral systolic murmur.
46	Mitral systolic murmur. Quality of first sound changes while listening to it.
54	Mitral systolic murmur.
60	" " "
61	" " "
72	Mitral systolic murmur.
74	Mitral systolic murmur, and aortic diastolic.
75	Mitral systolic murmur, and aortic diastolic.
77	Mitral systolic murmur.
80	" " "
82	" " "
184	" " "
125	" " "
129	" " "
130	" " "
133	" " "
134	" " "
137	" " "

MITRAL SYSTOLIC MURMUR.

Case No.	
139	Mitral systolic murmur.
140	" " "
44	" " "

MITRAL PRESYSTOLIC MURMUR.

14	Mitral presystolic murmur.
71	" " "

RELAPSE AND RECRUDESCENCE.

18	Recrudescence. Was normal two days, then fever for six days.
26	Relapse. Was normal ten days, spots, and baths required.
60	Recrudescence. Was normal four days.
77	Recrudescence. Was normal five days, then fever for six days.
119	Recrudescence. Was normal nine days, then fever for eight days.
112	Recrudescence. Was normal ten days, ate cabbage at seashore.
130	Recrudescence. Was normal six days, then fever for ten days.

PHLEGMASIA.

18	Phlegmasia. ?
31	Phlegmasia. Left leg; developed 28th day; no murmur.
74	Phlegmasia. ? Mitral and aortic regurgitation.
95	Phlegmasia. Left, developed 18th day; five days later a mitral regurgitation murmur is noted.
137	Phlegmasia. Both legs, less severe in right; developed 35th day; Mitral regurgitation.
139	Phlegmasia. Right, developed 15th day: age 27; rigid radials; orchitis.

HEMORRHAGE FROM THE BOWELS.

12	Hemorrhage from the bowels. No. 2, amount 8 ounces.
52	Hemorrhage from the bowels. No. 5, amount 63 ounces; peforation; died.
98	Hemorrhage from the bowels. No. 5, amount 64 ounces.
93	Hemorrhage from the bowels. No. 8, amount 84 ounces.

FURUNCULOSIS.

Case No.		
18	Furunculosis.	
19	"	Cervical glands large and tender.
26	"	Most numerous on face.
31	"	
37	"	Most numerous on hands.
40	"	Most numerous on back.
46	Furunculosis.	Most numerous on axilla.
48	"	Most numerous on trunk.
55	"	Acne in type.
81	"	
82	"	Acne in type, most on tibiæ.
84	"	Acne in type, most numerous on abdomen.
118	"	
125	"	

GLANDULAR INVOLVEMENT.

31	Mammitis.

LIVER.

60	Liver.	Acute hepatitis, jaundice.
86	"	Acute hepatitis.
49	"	Jaundice.

PAROTITIS.

72	Parotitis.

ORCHITIS.

75	Orchitis.
139	"

EYE.

138	Eye.	Acute cat. conj.

EAR.

70	Ear.	Deafness.
141	Ear.	Suppurative otitis.

BOWELS.

129	Bowels.	Ischio-rectal abscess.

SCURVY.

47	Scurvy.	Gums receding, spongy and bleeding.		
53	"	"	"	"
77	"	"	"	"
84	"	"	"	"

TYPHOID-FEVER PATIENTS CONVALESCENT WHEN RECEIVED. Thirty patients from Porto Rico gave a clear history of symptoms indicative of or a diagnosis of typhoid. Of this number 6 gave the Widal reaction. The record does not show the exact number tested, but most of them were not tried for the reaction. One had jaundice as a complication; another gave a history of three relapses, and 7 had chills as a complication. Two of them had spots when admitted to the hospital. In addition to these there were 3 convalescent typhoids received from Camp Meade.

This makes a total of 33 cases that very certainly had typhoid

fever, but who were so far recovered from it when they came under my care as to make them unsuited to admission to the class from which the statistics offered have been drawn.

Gangrenous Dermatitis. This unusual complication of typhoid fever was present in a number of the cases reported in this paper. I have already made a preliminary report of three of these cases in the *Philadelphia Medical Journal*, October 15, 1898. A partial history of the cases where gangrene developed is as follows :

Case I. (Hospital No. 1159, and No. 1 in this series).—S. M., aged twenty-two years. Camp at Fernandina, Fla. Patient walked aboard the hospital train, carrying the weight of his equipment, with the exception of his gun. The temperature was 105° F. at this time, and three hours later it was 106.4° F. The temperature continued between 104° and 105° F. for four days, and then ranged about 103° F. and over. His mind was cloudy at the time when there was not positive delirium, so that no history of his illness could be obtained. The spleen was enlarged, but no spots were present. One week after admission spots appeared, and the mind had cleared. The muscular sound of the heart was very deficient. The temperature was fluctuating between 100° and 103° F. He had a chill, and the temperature rose to 107° F. on the ninth day after admission; the respirations were from 40 to 52; there were crepitant râles at the lower lobe of the right lung, and the muscular sound of the heart was absent, and the second sound followed very quickly upon the first.

At this time there was noted a bed-sore and numerous boils.

On the tenth day the following note was made: On the right side, seventh interspace back of the anterior axillary line, a small furuncle appeared last night. Its greatest diameter was one-half inch. This morning there is necrosis of the integument, two by two and one-half inches. Underlying skin appears necrosed, with a central zone of darker color. Two inches below this, and more posterior, is a "sore" similar in character to that noted as present last night. It has a diameter of three-eighths of an inch. There is ecchymosis of the skin around the necrotic areas.

Eleventh day. Has abscess on inner side of left arm, one-third the length of the humerus. Another abscess over inner condyle of right arm. It was opened and contained foul-smelling pus. On buttocks there are multiple furuncles, closely placed and circumscribed, and show a tendency to form crusts. On left malar bone there is a rapidly developed necrotic area about the size of a half-dollar, and similar areas behind each ear.

Twelfth day. Feet cyanosed; delirious; between A.M. and P.M. skin on neck and over lower jaw softened over an area of one and one-half by two inches. It almost seems to melt while examining the patient.

Thirteenth day. Necrotic areas deeper. Patient emaciated to the last degree. Died.

Autopsy by Dr. Muerleck forty-five hours after death. Pronounced rigor. About five feet eleven inches. Necrotic areas as noted. Section: Tissues extraordinarily dry; knife creeks in cutting. Thoracic organs normal. Peritoneum markedly injected, recent adhesions. Liver extends one inch below floating ribs. Right lobe to median line; left lobe and lobus spigela markedly enlarged, extending around anterior surface of the spleen. Colon exceedingly distended, through peritoneal surface of which well-marked ulcers can be seen. Right kidney enlarged one-third, lobulated, distinct evidences of infarction. Left kidney almost double the normal size; capsule not adherent; marked infarction. Spleen enlarged, friable, lobulated, and infarcted. Post peritoneal glands extensively indurated. Extreme dryness of each tissue and organ.

Case II. (Hospital No. 1321, and No. 41 in this series).—F. M., aged twenty years. Admitted September 11, 1898. Ill for about a week. No spots; spleen enlarged; abdomen tympanitic.

13th. Active delirium. Second heart sound accentuated. Bronchitis with moist râles.

15th. Superficial cyanosis over trunk. First sound of heart weaker in force, second sound accentuated. Dermatitis about spine of scapula.

16th. The tissues show a decided tendency to break down. The skin above right ear and on left forearm have broken down. Although the temperature is approaching normal, the patient remains stuporous. Patient very ill; stuporous; respirations labored. Dermatitis on scapula is scaling. There is an area two by one inch of melted skin on forehead. Yesterday there appeared on either side of the sternum two patches of dermatitis, which rapidly attained the size of a silver dollar, and to-day are confluent. Result: Death.

Autopsy by Dr. Muerleck. Body of a male about five feet seven inches tall, fairly well nourished. Rigor mortis marked. Necrotic areas on neck and trunk varying in size from that of a pea to the size of the hand. These are located notably over the ulnar surface of the forearm, anterior aspect of the chest, epigastrium, ilcum, trochanters, also over forehead, zygomatic process, right ear, on back and sacrum, and on scrotum. Tissue moist on section. Heart normal. Liver enlarged, notably the right lobe; surface smooth. Parenchyma moist, and shows a degree of fatty degeneration. Spleen two and one-half times normal size, markedly lobulated. Substance friable. Peritoneum very much injected. No adhesions. Stomach distended. The mucous membrane is the seat of numerous ecchymotic spots or areas of dark red color. The organ contains considerable blood, altered by digestion. Right kidney twice the normal size, and shows traces of lobulation; capsule adherent, but strips easily. Cortical substance well defined. Lower pole of kidney is the seat of a hemorrhagic infarct, at the site of which the tissues are necrotic, forming a cavity the size of a hazelnut. The suprarenal body shows nothing abnormal. The same note is

true of the left kidney. The parenchyma of both kidneys shows cloudy swelling. Lungs: Left upper lobe is the seat of a hemorrhagic infarct, and the right upper lobe shows the same condition. Intestines: The ileum for a distance of from three to four feet from the ileocæcal valve shows very numerous typhoidal ulcers, over which the peritoneum is in a state of extensive necrosis. Vermiform appendix is deeply injected.

Case III. (Hospital No. 1506, and No. 59 in this series).—C. T. R., aged twenty-one years. Admitted September 23, 1898. Ill three weeks, and in bed ten days. Spots present; diarrhœa; gums inflamed; fecal incontinence.

October 5th. Over sacrum there is a purpuric area one by two inches. On left arm there is a similar patch the size of a half-dollar, and on left side of trunk there are six patches, varying in size from a dime to a nickel. These appeared as vesicles, and show a tendency to dry up. In the left axilla there is a small vesicle on a base the size of a quarter. On the left arm there are two vesicles, containing a serous fluid, each the size of a dime. Below the right scapula there is a patch the shape of an oyster shell, having a diameter of two and one-half by three and one-half inches. Around the circumference of this area there is a loss of the superficial integument, and at the centre there is breaking down into the deeper layers of the skin. The remainder of the patch is deeply pigmented.

6th. (The next day). The entire surface of the oyster-shell shaped patch is now raw and bleeding. The integument at the centre, which was intact yesterday, is broken down to-day. Above the sore there is a patch one by three-quarter inches, oval in shape, with the superficial skin lost and the underlying layers deeply injected.

8th. Oyster-shell shaped area looks healthier, less beefy, and there is over its surface a layer of odorous pus.

12th. Patient improved, patches healing.

17th. Has icterus. Patches nearly healed. Entire recovery.

Case IV. (Hospital No. 1530, and No. 72 in this series).—W. H. B., aged twenty-seven years. Admitted September 23, 1898. Ill ten days before admission. On admission had spots, diarrhœa, tender over spleen, persistent cold extremities, distressing cough with blood-streaked sputum, bronchial breathing and dulness at the base of the right lung.

October 3d. Blurring of the first sound of the heart at the mitral area 6–7. Has parotitis.

8th. Over the right scapula there is an area of erythema three by four inches, with glazed skin and breaking down of the superficial layers of the skin, and showing the papilla very prominently.

9th. On the posterior surface of the right thigh there is an oval-shaped patch as large as a half-dollar, with a dry, sealy condition of the epidermis.

12th. Both patches healing rapidly.

13th. Complains of toes being very painful

18*th*. Sores and toes well. There has developed a swelling to the left of the median line, midway between the umbilicus and the pubis, and extends to the pubis. It is two and one-half inches wide, and is tender on pressure. It is not superficial, and yet it is not deep enough to be within the abdominal cavity.

25*th*. Swelling is cone-shaped, more superficial, and the skin is red. It feels like a patella in the abdominal wall. This condition disappeared without operation. Result: Recovery.

Case V. (Hospital No. 1534, and No. 76 in this series).—G. S., aged twenty-three years. Admitted September 23, 1898. Ill nine days; spots; splenic enlargement; and receding, unhealthy gums.

30*th*. First sound of the heart weaker at the mitral area, and the second accentuated.

October 5*th*. Bed-sore over sacrum. On right ala of nose there is a necrotic area one-half by one-quarter inch. Near sacrum there is an ulcerated point and an oval vesicle containing clear serum, having an area of three-quarter by one-and-a-half inches. There is a circular vesicle over left scapula, having a diameter of one inch. On right side of the posterior axillary line, at the angle of the scapula, there is a vesicle one and one-half by one and three-quarter inches, containing clear serum. Two inches posterior to this there is a hyperæmic area with a small vesicle at one part of it. Heart sounds are better.

8*th*. Patches have not extended, and are healing rapidly. On left ileum there is an oyster shell-shaped patch three by two inches, with deepest injection at the margins, while the skin at the centre of the patch is livid. No vesicles. Above and out from this there is a vesicle with dull red base, one and one-half by one inch. To the left of this point there is another vesicle of the same character one-half by one inch. Just back of the great trochanter there is a cicular patch, one inch in diameter, with a deep red base, and glazed surface, but no vesicles.

10*th*. Patches healing. One small, new vesicle, containing serum, has appeared.

12*th*. Sores have so nearly healed as to show only what appears to be a desquamating dermatitis.

17*th*. Phlebitis of left leg. Phlegmasia.

Case VI. (Hospital No. 1541, and No. 81 in this series).—F. Q., aged twenty-four years. Admitted September 23, 1898. Ill one week; chill; nose-bleed; spots, and enlarged spleen.

October 13*th*. Has slight furunculosis over right ileum, also two small areas of necrosis, with vesicles. Result: Recovery.

Case VII. (Hospital No. 1188, and No. 92 in this series).—L. S., aged nineteen years. Admitted August 29, 1898. Ill three weeks. Vertigo, spleen enlarged, cough, no spots.

September 3*d*. Did not give Widal reaction.

5th. Spots appeared. Muscular sound of the heart weak. Second sound accentuated.

12th. Bed-sore at sacro-lumbar articulation, also one over spine of left scapula. Furunculosis. Pneumonia of left lower lobe of lung.

15th. First sound of the heart exceedingly weak, second sound accentuated. Heart action irregular. Coma vigil.

21st. Somnolent. Says he feels well. The back is denuded of the superficial layers of the skin over about one-quarter of its surface. No patches on face, legs, or arms. Feet tender to touch and motion. Muscular sound of the heart very weak.

23d. There is tissue necrosis involving three-quarters of the entire surface of the back. On the spine of both scapulæ there are two deep necrotic areas, each the size of a quarter. The disease on the back has lessened, but there still remains marked discoloration around the newly formed skin. Over the lumbo-sacral articulation there is a bed-sore five by six inches, showing clearly the uncovered muscles. Patient much improved. Tenderness along femoral vein. Complains of numbness of the left foot. Radial pulse has no volume. The skin over the spine of the left scapula has broken down over an area one and one-half by two and one-half inches. There are several small patches of necrotic tissue around this area. The necrotic areas on the lateral aspect of right chest have practically healed. Marked brachial pulsation throughout its entire course. There is a condition of arterial sclerosis of the brachials and radials. Complains of numbness in the fingers. There is quite a deep furuncle below and to the outer side of the left knee.

October 5th. Gave Widal reaction.

12th. Improved. The brachial and radial arteries in the right and left arm are still more corded. Rolls under finger, and can scarcely be obliterated. The condition is most pronounced in the right arm. There is blurring of both heart sounds at the mitral area.

31st. Brachial arteries cannot be compressed, radials less markedly rigid.

November 7th. Phlegmasia of right leg. Result: Recovery.

Case VIII. (Hospital No. 1248, and No. 93 in this series).—H. S. (hospital steward), aged nineteen years. Admitted September 29, 1898. Ill five days. No symptoms of typhoid. Malaise and coryza.

7th. Temperature normal for four days; allowed to be up.

11th. Went driving, ate Switzer cheese, rye bread, and drank milk punch. Returned to hospital at 11 P.M. The next day the temperature was 105° F., and a relapse, extending from the 12th to 15th, began.

26th. Urethral discharge. Tender joints and painful glands in axilla. Heart sounds good.

October 2d. Right leg and foot swollen and tender. Femoral vein tense. Tender along left brachial nerve, and the axillary glands are enlarged and the skin over them is red. Right brachial is free. No heart murmur.

6th. Had a chill. Skin over insteps and about joints on the hands is blue.

10th. Pneumonia of left lower lobe of lung. Great tenderness when the abdomen is lightly percussed.

12th. Had four hemorrhages, losing about three pints of blood.

16th. No muscular elements to the heart sounds. Delirious hiccough, persistent vomiting. Has "sores" on back and legs. Had four hemorrhages. Lost two and one-quarter pints of blood.

20th. Pulse irregular and extremely weak. Has a large bed-sore on the back. The right foot is of a purplish color; looks almost gangrenous. There is a large bed-sore on the back of the right leg.

22d. Right foot and leg half way to the knee is of a purplish black color.

25th. Discoloration has disappeared, and sensation has returned as far as the ankle. There is maceration of the skin of the foot, and over the great toe and heel the skin is black.

26th. Patch of gangrene back of bend of right knee, one by three-quarter inch. Also one on outer aspect of the calf of leg, oval in shape, two by one and one-half inches. Skin over these areas dark and purplish, with a greenish-gray centre.

November 11th. No heart murmurs. Leg amputated at the middle by Dr. Ransley. Patient now under the care of Dr. J. P. C. Griffith.

Dr. Rhein reports the persistence of the brachial neuritis, and diminished or absence of power in the biceps, triceps, and deltoid muscles. Result: Recovery.

Case X. (Hospital No. 1432, and No. 121 in this series).—N. R., aged twenty-one years. Admitted September 15, 1898. Ill one week. Malaise, chilly, and coated tongue. No spots.

22d. Spots, tympany, and a loud systolic mitral murmur, which is transmitted to the axilla.

28th. Decided redness of the nose and face. Delirious. Unable to keep him in the bath the usual length of time, because of cyanosis. Bath, eight minutes.

October 3d. Ecchymotic areas have appeared on the right side of the trunk, between the nipple and the lower edge of the ribs. The skin over this area is broken. On the left side of the chest to the side of the sternum, extending to the anterior axillary line, at the eighth interspace, there is an ecchymotic area, also another on left side between ribs and trochanter. Posteriorly there is an area very deeply tinted, extending from the left iliac crest to the lumbo-sacral articulation, and to the trochanter—an area six by seven inches. No break in the skin or sign of bed-sore.

4th. Patch on right elbow two by two inches. Skin broken over an area equal to one cent. Other patches have not extended. Patch over liver has an area of broken-down integument the size of a cent. All these areas are very painful. The only furuncolosis is one on the nose and three on

the left index finger. Complains of great pain in the ball of the great toes. Color of patches is deeper.

5th. Over purpuric area on hip the skin is markedly goose-fleshed. Some furunculosis about the buttocks.

10th. Skin over areas noted is very dry and shrivelled. Result: Recovery.

Case X. (Hospital No. 1240, and No. 33 in this series).—W. M., aged twenty-five years. Admitted August 29, 1898. Ill three weeks. Splenic enlargement, iliac gurgling, and tenderness.

September 3d. Patient actively delirious, and has to be tied in bed. There are spots on the right shoulder that are of a purple color. Heart sounds good.

8th. Exceedingly restless, constantly turning in bed. Mitral regurgitation. Result: Died September 13, 1898.

A bacteriological examination of these cases was made by Dr. James Walsh, bacteriologist to the hospital, and was as follows:

Two inoculations taken from the unbroken vesicles, one on agar-agar and the other on egg-albumin, showed cultures of staphylococcus pyogenes albus and aureus, no other bacteria being found. These were grown on the different media and put through the different tests that would prove their identity.

A culture from an ulcer after the vesicle had broken down showed the staphyloeoccus pyogenes albus and aureus in large numbers, and, in addition large numbers of several indifferent bacteria, as coeci, diploeocci, etc., were found.

The inoculability of the disease is evidenced by the experience of one of the nurses. A swab used in cleansing one of the gangrenous patches came in contact with a place on her hand where the skin was broken, and she promptly developed a sore which yielded cultures identical to those secured from the gangrenous area.

The treatment of these necrotic areas consisted in cleansing them and applying acetanilid and bovinine. Internally, they got whiskey, strychnine, and hemaboloids.

I desire to acknowledge my indebtedness to Dr. J. F. Schamberg and L. Job Lane (a medical student in the University of Pennsylvania) for the photographs I am able to show you, and to Dr. E. D. Mitchell, Dr. W. B. Fetterman, and Dr. J. P. Wales, resident physicians at St. Agnes' Hospital, for valued assistance in the collection of the data contained in this paper.

The etiology of this condition, aside from the bacteriological finding, is of interest because of the associated grave anæmia, the development of the condition at a time when the patient had passed through the period of the greatest severity of the fever, the almost

absent or exceedingly feeble first sound of the heart in five of these cases; the occurrence of mitral regurgitation in two of the cases; the cyanosis of the skin of the extremities that preceded the necrosis in one instance, and the hyperæmia of the face in another instance; the blurring of the muscular sound of the heart and the accentuation of the second sound; the arterio-sclerosis of most pronounced type in a patient of nineteen years; the oval shape of several of the areas, and the subsequent development of phlegmasia, all lend weight to the assumption that the disease is of embolic or thrombotic origin. The post mortem findings—ecchymotic areas in the stomach, and infarction in the upper lobes of both lungs in one instance, and in the left kidney of the same case, and in the other instance that came to autopsy there was infarction of both kidneys and the spleen—all these facts point to the one exciting cause.

The Frequency of the Occurrence. Dr. W. W. Keen, in the Shattuck Lecture delivered June 6, 1896, says: "While gangrene is an important complication or sequel of typhoid, it is, fortunately, rare, so that most practitioners, even men of vast experience in large hospitals, have never seen a case; for example, Flint and Murchison. Holscher, in 2000 fatal cases of typhoid, does not report a single case, though he records 59 cases of thrombosis of the femoral vein, and Bettke in 1420 cases, found only four cases of gangrene, all limited to the toes."

My own experience is in marked contrast to this. In the cases reported the trunk was the most frequent seat of the disease, and the face, the arms, the thighs, and in two instances that terminated in death, the scrotum was involved. In only one case did the disease attack the foot, and while the patient survived the foot was lost.

It would have added interest to the search for a remote exciting cause had all of the patients exhibiting the disease come from the same camp; but such was not the case. Neither had the trouble confined itself to the patients in one ward or one floor of the hospital, nor to those under the care of any set of nurses.

DISCUSSION.

Dr. J. M. Da Costa: My experience has been much the same as that of those who have already addressed the College. The cases that I have had in the wards of the Pennsylvania Hospital as new cases, or seen soon after admission, numbered sixty-five. Of these, about thirty of very grave character were admitted at one time. Yet, medically speaking, they presented nothing but what I have noticed in typhoid fever elsewhere, and nothing but what I remember to have observed in the Civil War. Compared with ordinary practice, I ought to except the greater tendency to thrombosis of the leg. It is, indeed, a remarkable fact that among the cases lately met with in soldiers at the Pennsylvania Hospital, in 135 cases of typhoid fever that were seen early, or were transferred to me at a late stage, I should have had eighteen cases of thrombosis of the leg—in, therefore, 13⅓ per cent. It is also a remarkable fact that the majority of these cases were double; of those that were single, more affected the left leg than the right leg, for only in two was this alone the seat of the lesion. Looking at the reports of the other cases in the hospital, we found that we had in all about thirty cases of phlegmasia alba dolens in 215 typhoid fever patients—nearly 14 per cent. This is, I think, an extraordinary circumstance. I rarely go through a winter's term at the Pennsylvania Hospital without a case or two. But my own experience must be that of every Fellow present, that 1 or 2 per cent. of this complication is all that really happens in ordinary hospital practice.

Another point is with reference to the condition of the skin. There were two cases that presented symmetrical ecchymoses, in one almost exclusively confined to the ankles. This is a very rare complication. I do not mean simple blush, but ecchymotic spots. These two cases I examined closely for scurvy, both the gums and otherwise; but no signs of scorbutic taint were discernible, nor was there evidence of disease of the heart or the blood-vessels.

Of the sixty-five under my immediate control, four died—a little more than 6 per cent. Of the four deaths, one occurred a few days after admission to the ward in a man whose temperature, when he came from the hospital train, was over 106°; the second, after an intestinal hemorrhage followed by a severe pulmonary congestion; the third, from most extensive ulceration of the bowels, about one hundred typhoid ulcers, small and large, being found in the intestines, in which there was more diseased tissue than healthy tissue to be seen; the fourth, on the seventy-second day, in a man in whom the fever process had been long over, but whom it was impossible to nourish. He reached an extreme state of emaciation, was a mental wreck, the fatuity being well marked, and he died completely exhausted.

The first thirty cases, all admitted simultaneously, and among whom every one of the deaths happened, were of very grave character; how grave may be judged from the fact that twenty-eight of them were at the same time delirious. Nearly all came from Camp Meade. Among the recoveries were two in whom the temperature reached over 106°.

The treatment pursued was by betanaphthol, about three grains every four hours, by meeting individual symptoms, by keeping the temperature down with sponging, and, so far as possible, by not disturbing the patient oftener than every two hours. Following in civilians the same treatment, very nearly at the same time or a little later, in the general wards of the Hospital, I had, among sixty-one cases, six deaths—nearly 10 per cent.—the variation in age being rather greater. The soldiers, therefore, picked young men, in good health previously, showed, notwithstanding the gravity of most of the cases, a considerably less mortality than was found among those who generally seek relief in hospitals, and whose prior condition is not apt to be so good.

But, perhaps, the best contribution I can make to this instructive discussion is the proof of the not infrequent combination of malaria and typhoid fever. I have ten cases of typhoid fever to offer, proved such by unmistakable clinical features, and in nearly every instance by the Widal test, where the microscopical evidence of the malarial organism was found. In every case this was of the tertian type or the æstivo-autumnal type, generally the former; the activity of the amœboid movements is, in a number of the notes, specially commented on. I have the notes and the temperature-sheets of the cases in my hand, but it would be tedious to read them in detail. I will, rather, give you a general analysis, and point out what they demonstrate. They all have very much the same history. The cases may or may not have had malarial chills before the typhoid fever; the great majority had not had chills. They came, with one exception, from Camp Meade, near Harrisburg, the exception being a case from Porto Rico. But, on investigating the matter, I found that most of them had been sent to Camp Meade from Camp Alger, in Virginia, a highly insalubrious and malarial camp. It is nearly always a chill that calls attention to the malarial complication. The peculiarity of these chills is that they do not happen early in the fever malady; indeed, in only one instance of the ten was such the case. They come on late, and sometimes not until a relapse. The chill is associated with, or rapidly followed by, marked temperature rise and by sweating. It is important to note, however, that we have these without a chill, and only detect their meaning by finding the malarial corpuscle. Another peculiarity of these mixed cases—and it was demonstrated in every one of the ten—is their long duration; as a rule, they pass beyond the fourth week. In one instance the malarial corpuscle was found where there had been no chill and no marked or abrupt alteration of temperature in the long protracted case.

As the chills are the most striking manifestation of these mixed cases, I will state in detail how they happened in some of the ten cases to which I am now calling your attention, a few of which had been originally under the care of my colleague, Dr. Meigs, who kindly permits me to mention them. In one patient, H. S., the first chill happened on the twenty-sixth day of the disease, the temperature rose to 103.6°, fell by the next day to normal, and rose again in the succeeding twenty-four hours to 103.6°. No malarial organisms were detected on examining the blood. The subsequent day he had two chills, in the first of which the temperature rose to 105.8°, in the second to 104.6°. The chills lasted an hour. After the second one the blood was examined, and a number of tertian plasmodia were found in active form, all intracorpuscular, and several pigmented. Placed on quinine, no more chills happened, and no organisms were to be seen in the blood. The convalescence continued uninterrupted except for a phlegmasia dolens of the left leg.

In the case of J. T., late in the disease, rises of temperature occurred, from normal to 100.8°, without chills; the malarial organisms of tertian type were found during these fever rises. In another case, F. S., there had been chills before admission to the hospital, but it is not certain that he had any before his attack of typhoid fever. Five days after admission he had a severe chill lasting two hours, during which the temperature rose to 106°. A microscopical examination of the blood showed the tertian form of malarial corpuscles in unusual numbers. In another case, I. L., the chill happened on the forty-seventh day of the disease, and lasted nearly an hour, the temperature rising from 99° to 105°; the fever was followed by profuse sweating and a drop of the temperature to 99°. The type of plasmodium of malaria was not well defined.

As a striking illustration of late appearance, I may cite the case of M. B., whose remarkable and extended temperature-sheet I now show you. You will see on it three distinct relapses, and it is a most curious circumstance that it was only in the third relapse, after the occurrence of a violent chill, that malarial organisms were found. This chill took place on the eighty-eighth day of the disease. You may be interested to know, further, that this patient had various complications, such as otitis media, stiffness of the muscles of the neck, and swelling of the knee. Reflecting on this and on the extraordinary duration of the case, and bearing in mind the remarkable observations recently made by Dr. Musser and Dr. Sailer on "Malta Fever," I had the blood examined for this disease. Dr. Thomas S. Kirkbride and Dr. Kneass most kindly undertook the investigation, but found Malta fever not to exist, while, repeating the Widal test, they got unmistakable evidence of typhoid fever.

We see, then, that these cases of mixed infection are distinguished not only by chills, but by long duration of the febrile malady; marked irregularities of temperature are also very suggestive of the coexistence of the

morbid states. We ought, therefore, to examine for malarial corpuscles in every case of typhoid fever, not only where chills occur, but also where the fever is prolonged or irregular, or where there are frequent relapses. Moreover, some of the cases whose records I have here analyzed prove that we may not always at the first examination obtain conclusive microscopic evidence. The coexistence of the two poisons is very strange; but the slow manifestation of the malarial symptoms shows the typhoid fever poison to be the overpowering one, and the malaria is for the time checked.

Cases such as I have referred to, even cases merely suspected in which the fever persists for a long period, suggest the free use of quinine. From sixteen to twenty grains of quinine daily, given for a few days, was generally found sufficient to break up the malarial complication; smaller doses were then continued until full convalescence. But in some cases much larger doses than those mentioned are required, or quinine is found to fail entirely. Thus, in a case seen with Dr. Potter in Germantown, in which the double infection was contracted in Porto Rico, over thirty grains were given for days without any marked impression being produced. The patient only recovered when Warburg's tincture was taken in drachm doses three or four times daily. In some of the protracted cases quinine was used hypodermatically with benefit, given in the shape of the bimuriate of quinine and urea, in ten-grain doses every twenty-four hours until the temperature became normal.

Dr. J. M. Anders: I gathered from the paper of Dr. Tyson, and more especially from that of Dr. Meigs, that the cases of typhoid fever among soldiers under their observation ran a comparatively mild course. My own experience coincides more nearly with that of Dr. Da Costa, who met with cases of a serious type. According to my observation, the severity of the disease depended very much upon the camp from which the patients were brought; thus, among 100 patients conveyed from Camp Alger to the Medico-Chirurgical College Hospital, most of them suffering from typhoid fever, many presented a serious form of the disease. Again, of 100 brought from Camp Meade, the majority of the cases presented a serious type of the affection, while complications were numerous and varied. Dr. Stahl stated that hyperpyrexia was present in some of his cases. Of the totality of 266 cases treated at our hospital, twenty-six showed hyperpyrexia; it should be remarked, however, that circulatory depression and the nervous symptoms were quite as well marked as in other cases showing less fever. Two cases developed insanity during convalescence; one, after he had left his bed.

Dr. Stahl referred, I think, to the fact that the first sound of the heart was weak in some of his cases. We noted this condition in twenty out of our 266 cases, and three finally terminated fatally amid the signs and symptoms of systemic collapse. He also spoke of an irregular action of the heart. This was noted in a large percentage of the cases brought to the Medico-Chirurgical Hospital, and while brachycardia is an almost invari-

able symptom during convalescence from typhoid, it occurred in three of our cases during the height of the disease. Two of these recovered, the third died.

Reference has been made to intestinal hemorrhage—a symptom that occurred in more than the usual proportion of cases. This I account for chiefly by the fact that these men were conveyed for long distances from the camps to the various hospitals, and were subjected not only to rough motion on trains and wagons, but to considerable handling as well. In five cases that terminated fatally at the Medico-Chirurgical Hospital, death was preceded by copious and repeated intestinal hemorrhage.

In this connection the question arises, What was the effect of the removal of these soldiers from the camps to the hospitals upon the subsequent course of the affection? From the mortality-rate as given here to-night and from the mortality-rate at the Medico-Chirurgical Hospital, (13 out of 266 cases), it seems to me that it was not unfavorable, if we except the somewhat greater tendency to hemorrhage of the bowels and, possibly, also to collapse of the circulation.

Pulmonary complications have also been present, with a somewhat increased percentage of cases of acute bronchitis and lobular pneumonia. Hæmoptysis developed in two cases under my care in the second and third weeks of the disease respectively; it was unassociated with any other symptoms, save, perhaps, a little more than the ordinary extent of the usual bronchitis. There was no family or personal predisposition to tuberculosis in either case, and no tubercular lesions developed subsequently; they both recovered.

In one case jaundice was met with, and it was present when the case was admitted. Dr. Da Costa, in an article recently published, has called attention to this as a rare symptom in typhoid fever, and states that it is dependent upon a variety of causes; he has also pointed out that it appears either in the middle period or not until late in the fever. In the case that was admitted to our hospital, it came on about the end of the first or the beginning of the second week. It continued until convalescence was established, and then gradually disappeared.

Two cases of purpura were also noted in the cases under treatment at the Medico-Chirurgical Hospital. In one of these the purpuric spots were confined to the abdomen, and in the other to the abdomen and legs. One case recovered; the other, in which the spots were confined to the abdomen, died, and the immediate cause of death was, I think, intestinal hemorrhage.

Three cases of yellow palms and soles were also met with, coming on early and remaining until convalescence was well established, as is usual in such cases.

The Widal reaction was referred to by all of the essayists of the evening, and there was shown a considerable diversity of opinion as to its practical value. I feel convinced that the Widal reaction enables us to make an

assured diagnosis in the majority of cases at an earlier period than can be done from the symptoms and the peculiar course, including the characteristic eruption, since it cannot be said that the first crop of eruption settles absolutely the diagnosis. In ten of the cases at the Medico-Chirurgical Hospital a negative report was constantly returned from Prof. MacFarland's laboratory, though the symptoms and course pointed strongly to the existence of typhoid fever. In a considerable percentage of the cases, perhaps 25 per cent., the reaction first appeared after the symptoms and successive crops of eruption had cleared the diagnosis.

I was very much interested to hear that some of the cases treated in the University, Pennsylvania and other hospitals showed the presence of the malarial plasmodium in the blood. In about one dozen cases at the Medico-Chirurgical Hospital the temperature-curve, occurrence of chills, and decidedly enlarged spleen pointed strongly to the probable existence of a dual infection, but a careful microscopical examination of the blood, and in most of the cases, repeated examinations of the blood by Prof. MacFarland, failed to reveal the plasmodium in a single instance. Surely these results specially emphasize the fact that in the presence of an undoubted case of typhoid fever, the occurrence of chills and an intermittent fever-curve is always insufficient for the diagnosis of typho-malarial fever.

Thirteen deaths occurred at the Medico-Chirurgical Hospital out of the total number, or 266 cases.

With reference to treatment, apart from stimulation, careful feeding, and hydrotherapy, the best results were obtained from the use of normal saline solution in intestinal hemorrhages. In one case in which death seemed imminent, intravenous injections of the saline solution by Profs. Hughes and MacFarland led to recovery, and in one of my own cases a saline infusion by hypodermoclysis was followed by recovery after the patient had been considered moribund. With reference to treatment of intercurrent lobular pneumonia and other secondary infectious inflammations, I would like to emphasize the importance of energetic and methodic alimentation, if begun early. If the cases are first seen at a later stage, then active stimulation is, perhaps, better than feeding.

Dr. H. A. Hare: I have very little to say with reference to this subject, because I was out of town when the greatest number of patients were brought to the Jefferson Hospital, and I asked Dr. S. Solis Cohen to take charge of the wards. He can give the College a better account of the cases treated than I, and I hope you will give me permission to ask Dr. Cohen to speak in my stead.

Permit me, however, to say a word or two briefly with regard to the question of malarial complication. In two papers, one by Ewing, of New York, and the other by Neuman, reference was made to careful examinations of the blood for the plasmodium, and both writers agree that when the plasmodium was present it was rather put aside by the typhoidal infection, and

then as the typhoidal infection passed away, so to speak, the signs of malarial infection came on again with increased vigor. Therefore, their results would seem to confirm the opinion expressed by Dr. Da Costa, that the malarial infection definitely prolongs the febrile condition, and that this complication should make us suspect malaria.

Another point with reference to the excessive doses of quinine which Dr. Da Costa says are necessary. This is probably due to the fact that the stomach does not absorb the medicines well in typhoid fever. Small doses will be efficient if given hypodermatically or intravenously.

One other point of interest is with reference to the mortality according to the weeks of illness on admission. It seems to me we get a better idea of the mortality of the disease if instead of saying 100 were admitted and so many died we note how many were admitted: how many in the first week of illness, how many in the second, how many in the third.

Finally, a point which impressed itself upon my mind on the hospital train coming down from Camp Meade was the undesirability of the woven-wire mattress. On three occasions I had to send a messenger to the engineer to ask that he would run the train at a lower rate of speed. These woven-wire spring mattresses tossed the patients up and down, and I was obliged to keep the nurses sitting on the sickest patients to keep them from being bounced out on the floor. A hard mattress upon a plank would have been much more comfortable than woven-wire mattresses, and their use was a mistaken kindness.

Dr. Alfred Stengel: I would like to refer to only two or three points. The cases which I treated during the summer have been alluded to by Dr. Tyson. With regard to the question of malaria, my experience has been confined to two cases in which the hæmatozoa were discovered. During the early part of the coming of the soldiers to the hospital we examined every patient's blood for the malarial parasite, whether there were symptoms indicative of malaria or not, and never found any organisms during the febrile attacks. In two cases that came under my own observation the tertian organisms were found during convalescence. One patient had been discharged from the hospital, but returned with chills and a very large spleen, and had organisms in his blood. In the other case the manifestations were not so pronounced, but there was distinct tertian malaria during the convalescence. There was no question about there being two infections running side by side in either of these cases; there was simply one affection (malaria) in abeyance, while the other (typhoid fever) ran its course as if the malaria was not there.

A point that is of great importance, I believe, in the study of these malarial cases was brought home to me the other day, when I had the pleasure of seeing some preparations made by Dr. Ewing, of New York. In one case there were very few organisms to be found in the blood, and these were non-pigmented, and, therefore, more easy to be overlooked. After death

the various tissues were cut, and the specimens showed immense numbers of organisms. The thought was suggested to me how easily the malaria might be overlooked, particularly in the examinations we make during the course of the typhoid fever, and I should be less likely now to assert that malarial organism was absent in the blood examined during the course of the typhoid fever.

One of the cases which came under my treatment had jaundice; and this, while it must be put down as jaundice occurring in a case of typhoid fever, had nothing whatever to do with the typhoid fever. It was due to cardiac disease and consequent hepatic congestion.

With regard to the Widal test, the statistics of the cases which I saw were not as favorable as the statistics of the Widal reaction have been previously in my experience. I believe this is due to technique rather than to the deficiency of the test. I am still inclined to believe strongly in the test.

Dr. S Solis Cohen: I had not expected to take part in the discussion, and have not provided myself with statistics. I can speak only of general impressions imperfectly remembered.

The most notable fact I now recall concerning the cases of typhoid fever that I saw at the Jefferson Medical College Hospital was the asthenia of the patients. Alike, those who arrived early in the disease, or late in its course, or in the beginning of convalescence, seemed to be prostrated out of all proportion to the severity of the symptoms that they manifested or of which they gave an account. One young fellow insisted that he was well when admitted; he had tramped many miles to the train, carrying his pack. He was kept in bed simply to watch him, and on the third day began to show symptoms of typhoid fever, the spots appearing seven days later. The complications that most impressed themselves upon me were parotiditis and orchitis. In one of the cases of orchitis there was found later tuberculosis of the testicle, and in one or two cases there was suspicion or history of venereal disease preceding the typhoid. The blue spots were very marked in two or three cases, pedicati being absent, and in one case so clearly hemorrhagic—purpuric—that I had a painting made of them for record. This case also showed malarial organisms. Every case coming into the hospital was examined for malarial organisms and for the Widal reaction, and a blood-count was made. I hope the records will be available for insertion in the printed report of this discussion. Many cases of mixed infection were found. The occurrence of chills in the cases proving to be typhoid fever only was not common, although if I recollect aright a number exhibited an intermittent temperature-course, notwithstanding the absence of the malarial organism upon repeated examinations.

I do not recall failure of positive Widal reaction in more than one instance of typhoid fever; but in many cases that proved not to be typhoid it gave a positive response also. My general impression con-

cerning the Widal reaction is that if I had made a positive diagnosis of typhoid fever on the general aspect of the case a positive result from the Widal reaction would not cause me to change that diagnosis.

Concerning the eruption in typhoid fever, one case exhibited profusely scattered spots, very coarse in character; and some patients having come in with measles, and others having given history of having had measles or having been exposed to it, the possibility of measles was entertained in this case; but the general symptoms did not develop, nor was the rash characteristic, and it seemed to be simply a pronounced variety of the rose spots. I have seen two other cases quite similar—one recently at the Philadelphia Hospital. In all the rash occupied the trunk and extremities, but the face was not affected.

We had during my term of service at the Jefferson Hospital but two deaths among the typhoid cases; what the total number of cases was I do not remember, but will endeavor to find from the record. In one of these cases the patient was admitted after having had several hemorrhages from the bowel and with numerous abscesses over the body. Dr. J. Chalmers Da Costa saw this case and operated upon some of the accessible abscesses, notably in the cellular tissue of the hand and arm. The hemorrhages had ceased before the patient's death. He was treated by hypodermoclysis and oxygen inhalations, and other means were employed to keep him alive, but without success. I may interpolate here the account of another and in some respects a remarkable case of intestinal hemorrhage. The man had had several hemorrhages on the train coming from Tampa, and was admitted in a state of profound exhaustion. We were expecting him to die very shortly, but to our surprise he recovered. This man had a very large and deep decubitus ulcer, which seemed to be much benefited by the use of formalin-gelatin in powder. One point in the treatment of hemorrhage that I learned from this case was to wash out the bowel, as Dr. McCormick, of Williamsport, advises. We first tried to prevent the bowels from moving, but despite treatment a number of hemorrhages took place. We then washed out the colon, removing a number of clots and some thickened blood, and the hemorrhages ceased. We used hot water, however, and not ice-water, as Dr. McCormick does, and we continued to keep the patient thoroughly morphinized and to administer the least quantity of water consistent with comfort. Food consisted of expressed beef-juice, of which the patient received but half an ounce at first and later two ounces every second or third hour.

The second fatal case was one of perforation in a man who had come in on the tenth or twelfth day of the disease in bad condition. He constantly complained of pain, which seemed to be referable to the right kidney or ureter rather than to the intestine. Dr. J. Chalmers Da Costa, who was called in consultation, agreed with me in this supposition, and as the urine suddenly became very scanty we thought that in some way there might

have been a twisting of the ureter. Later the patient went into collapse, in which condition he was seen, in my absence from the city, by Dr. Salinger, who diagnosticated perforation. When I saw him a local peritonitis was evident. The patient rallied and lingered for two or three days after this, causing us again to doubt the diagnosis of perforation. Operation was deemed inadvisable by Dr. Hearn and Dr. Da Costa as well as by me. The autopsy showed perforation, but also showed extensive calculous disease of the right kidney, with a large number of abscesses; thus the confusing symptoms seemed to be accounted for.

The treatment used most largely by myself and by my colleague, Dr. Salinger, under whose more immediate care I placed a number of the patients when the total became too great for a single attendant, was largely the cold bath, though, in my personal cases, with certain modifications of the vigorous Brand system. The rule that I have adopted in giving the cold bath in typhoid fever since I first used it in 1887, and from which I have but rarely departed, is never to try to reduce the temperature more than one and a half or two degrees by a single bath, certainly not to the normal line. I look upon a chart in which the temperature see-saws up and down, sinking to the normal line and below, as an undesirable chart, inasmuch as it simulates a septic fever. It is a chart which nature never gives us in a case of typhoid progressing normally toward recovery, and which we should therefore not attempt to get artificially. Typhoid fever is a continued fever; we should not try to convert it into an intermittent fever. In giving the cold bath I have tried to adjust the temperature and frequency to the needs and susceptibilities of the individual case, gradually reducing the upper level of the temperature line rather than suddenly reducing the lower level, with return to the original high point. I believe that cases thus treated run a shorter course and are less liable to relapse, though I have not statistics large enough to prove this to the satisfaction of anyone else.

In addition, an ice-bag is kept constantly on the patient's abdomen, except when he is in the bath; often ice is kept with the same persistence to the head. When the temperature does not reach the bathing point —102.3° to 103° F.—sponging is employed. Some cases are simply sponged and not bathed. Dr. Salinger and I made a number of comparisons apparently in similar cases, he following the strict Brand method, and I must confess that his patients did about as well as mine.

Believing in what is roughly termed intestinal antisepsis, I gave drugs to act in that manner. Benzonaphtol, betanaphtol, betanaphtol-bismuth, bismuth salicylate, salol, and guaiacol carbonate were used, and no special advantage was noted of one drug over the other. In the obstinate hemorrhage referred to, silver nitrate and betanaphtol-bismuth were given together for a while. In cases seen sufficiently early calomel was given to empty the bowel. In some cases turpentine was given, and did the work expected of

it. Hydrochloric acid was generally used in convalescence. Cases with malarial complications received quinine, and urea hydrochlorate, either hypodermatically or by the mouth. Strychnine sulphate in small doses, $\frac{1}{100}$ grain every waking hour, was given, on account of the marked asthenia. Alcohol was used freely in some cases, and in others not at all. As a rule, much more alcohol was given than I am in the habit of using in typhoid fever.

Major Peyton: It is too late for me to take up your time. I noticed one point made—I think by Dr. Hare—in regard to the statistics, which impressed me very much. It is a fact, that in a great many instances it was utterly impossible to know or find out at what stage of the disease the cases arrived in the hospitals in Philadelphia. In a great many cases there was absolutely no record, nothing at all to show even a diagnosis, much less the length of time the case had been sick. I have seen the one side of the work and you gentlemen have seen the other side.

Dr. A. C. Abbott: Notwithstanding the lack of confidence that some of the gentlemen have expressed this evening in the value of the Widal test for typhoid fever, permit me to say that when it is applied on so large a scale as we see it in the Municipal Laboratory of this city the results are fairly satisfactory. During the past twelve months the test has been applied to samples of blood from 4597 suspicious febrile conditions. Of these 3060 were civilian cases and 1450 were from among soldiers brought to the hospitals of this city. The results of our examinations in the group of civilian cases agreed with the diagnosis of the attending physicians 4366 times—that is to say, there was a discrepancy of 2.8 per cent. On the other hand, the discrepancy that occurred between the results of the test and the clinical diagnosis in the cases among the soldiers was very much higher, being approximately 10 per cent. These figures do not represent fully the work done by the laboratory, for the reason that in a certain proportion of cases it was impossible for us to get subsequent data from the physicians in charge, and we have been obliged, therefore, to leave these cases out of our statistics. On the whole, however, I am satisfied that the test must be regarded as of assistance in the diagnosis of typhoid fever.

Dr. Da Costa, in closing, said: The objection, in my opinion, to giving quinine in typhoid fever hypodermatically is, irrespective of the pain it may occasion, the risk of abscess. The state of the blood and the low vitality of the patient predispose to this, and I should always prefer not to give quinine hypodermatically so long as there is likelihood of its being of real use in any other way.

Dr. S. Solis Cohen: I was very much gratified to hear two things mentioned by Prof. Da Costa as having come under his personal experience.

One is concerning the occurrence of the malarial organism in connection with typhoid fever. When at the Pan-American Medical Congress in Washington I contended for this explanation of certain cases it was not well received. Since then the observation has been repeatedly confirmed. Secondly, I was glad to hear Prof. Da Costa commend the hypodermatic use of quinine and urea hydrochlorate in the treatment of the malarial complications of typhoid fever. Since 1884 I have been almost vainly trying to get the profession to adopt this drug, which I consider the best quinine preparation to use in acute malarial fevers as well as in obstinate and chronic cases. With care no bad local effects need occur.

In relation to the occurrence of the malarial organism in typhoid fever and its disappearance during the fever, I may here recall that in a clinical lecture at the Philadelphia Hospital, with exhibition of the patient, some two or three years ago, I took occasion to report the following experience, namely, that the organism was found on the admission of the patient with chills and fever. After admission the symptoms of typhoid fever developed, and the case ran the typical course of typhoid fever, the malarial organism never being found during that period. With the cessation of the typhoid symptoms it was again demonstrable. What it is that makes it difficult to find the organism during the course of typhoid I do not know. I do not know whether it is veritable absence or merely lack of discovery. It may be that the typhoid toxin has a destructive effect upon the forms of the organism usually recognized. As the same observers made all the examinations in my case referred to, the question of skill alone does not offer an explanation.

Dr. Joseph Sailer: I think it only fair to the Board of Health to state that during the last three months at the Presbyterian Hospital, where I had an opportunity of seeing the results of the Widal test, the blood from about sixty cases of typhoid fever was sent to the city laboratory with a positive return in every instance. In another case, a persistent fever of very obscure nature, specimens of the blood were sent to the laboratory on four different occasions, and on every occasion the report was negative. I then tested the blood myself by the Widal reaction, and obtained a negative reaction. The subsequent clinical history justified the exclusion of typhoid fever. In a number of other cases supposed to be typhoid when admitted to the hospital the report was negative, and in every instance the subsequent course of the case apparently confirmed it.

Dr. Arthur V. Meigs, in closing the discussion, said: Dr. Anders misunderstood me; I did not say that the cases of typhoid fever of which I had charge were mild; a great many of them were very severe.

One word with regard to the Widal test. Perhaps what I have said may be misunderstood. What I intended to express is that I have come to the

conclusion that the test as applied by the authorities of the Board of Health is unreliable as a diagnostic aid. I have had no opportunity of finally judging in regard to the merits of the test. There are some bacteriologists who say it is not reliable if made from dry blood, but that serum should always be used. I wish to say that I have a great respect for the scientific work done by the Board of Health, and I have always instructed my assistants to send no blood to the laboratory except that of persons suspected to have typhoid fever. I have not been able in all cases to prevent the playing of tricks in sending blood from persons suffering with various diseases, or even of those in good health. The efforts of the health authorities to carry on their scientific work should be treated with respect, and they should be fairly and openly dealt with.

THE RÖNTGEN RAY DIAGNOSIS OF RENAL CALCULUS.

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Although our knowledge of the etiology and symptomatology of renal disease has made great advances within recent years, there are many cases in which an absolute diagnosis is practically impossible. Those who have been called upon most frequently to make a differential diagnosis admit most freely that often they cannot establish a diagnosis because of the indefiniteness of the symptoms.

The classical symptoms of calculous nephritis are not always present, and when apparently present operation has frequently failed in detecting a calculus. The cases that are most easily diagnosed have passed calculi at some previous time, while repeated urinary analyses show crystals of uric acid or calcium oxalate, with an intermingling of blood, at times in large quantities, in microscopic quantities all the time. Renal colic has manifested itself in repeated attacks, while marked tenderness and constant pain are persistent symptoms that are aggravated by violent exertion or a sudden jarring.

These symptoms are not always present, nor can the diagnosis be made with ease in all cases. As Mr. Henry Morris has said, in his valuable Hunterian Lectures of last year, "in many cases, however, mistakes in diagnosis are bound to arise. Morbid conditions other than stone will be found from time to time in which the character of the pain and the state of the urine point to calculus as the probable cause."

The ease with which the symptoms of the different morbid conditions of the kidney may be confounded with calculous disease will be seen by glancing at their symptomatology, while the masking or absence of a symptom must increase the difficulty. Thus, in the early development of malignant or adenomatous disease, before the tumor can be detected, the clinical symptoms are pain referred to the diseased kidney, with a recurring hæmaturia that is slight, and yet has all the characteristics of calculous disease.

In the milder cases of hemorrhagic pyelitis red and white blood-cells are present in the urine. In the more severe forms the hemorrhage is more marked, while the pain varies in intensity and is referred to one kidney. Operation in many of those cases suspected of being calculous disease has shown that no calculi were present. The tissues of the kidney were not altered. The only pathological change noted was a thickening of the mucous membrane of the pelvis, and calices, with an infiltration of cells in its stroma.

The presence of blood, pus, epithelium, and shreds of tissue are common to both calculous nephritis and tubercular nephritis, while the symptoms of pain and tenderness on pressure in the area of the kidney vary in both from time to time. The only differential diagnostic point that is of real value is the presence of tubercular bacilli in the urine. Their detection and recognition are often matters of great difficulty, and are sometimes impossible. In many other forms of pyelonephritis the symptoms are often so similar to those of calculous disease as to be very confusing.

In cases of movable kidney we may have severe paroxysms, characterized by abdominal pain, chills, nausea, vomiting, fever, and collapse. Blood and pus may be present in the urine. When the amount of mobility is slight the similarity of the symptomatology makes the differential diagnosis very difficult. We know, too, that the two conditions may exist in the same kidney at the same time, which adds to the complications. Mr. Morris says of such cases: "I have explored several cases in which the diagnosis was very doubtful, but the severity of the suffering was so intense that the patients have willingly submitted to an operation. The exploration has proved the absence of a calculus, but has revealed a degree of

mobility behind the peritoneum, in what I have designated a cinder-sifting manner, altogether out of proportion to any mobility detectable by clinical examination; in some of them no mobility whatever could be made out by bimanual abdominal palpation. In these cases complete relief has been obtained by nephropexy. In other cases I have found, instead of calculus, the kidney more or less displaced, and though little, if at all, movable, yet greatly engorged with venous blood, owing to distortion of the veins at the hilum. In other cases, again, the kidney has been swollen and congested with venous blood obstructed by the pressure of tough and condensed perinephric cellular tissue."

There are many other conditions depending on a certain amount of mobility in the kidney, as where the ureter is sharply bent, that may produce many symptoms similar to calculous nephritis.

In ureteritis we have a condition of which very little is known either of its pathology or symptomatology. If we judge from the symptoms produced by inflammation in other portions of the urinary tract, it is capable of producing symptoms and conditions that would simulate renal lithiasis. When its pathology and symptomatology are more clearly determined we shall probably find that it will account for many of the cases that are now classified as nephralgia, or that are operated upon for calculi where none are found.

A case that may have been of this character, and that illustrates the impossibility of excluding calculus by ordinary diagnostic methods, was reported to the French Academy of Surgery by Le Dentu. The patient had had for twelve years what were apparently attacks of nephritic colic. The only symptom that was wanting was the presence of crystals of uric acid or other acids in the urine. These crises were frequent during two or three years, and were modified by the use of mineral waters. After a period of apparent cure they recurred, and had persisted during the past six years. It was during a particularly severe attack that it was considered best to make an exploratory nephrotomy, the symptoms seeming to indicate the presence of a calculus. None, however, was found, although the kidney presented marked external appearances of inflammation. This author concludes that there are cases, purely medical in their nature, that so closely simulate calculous disease as to make the

diagnosis impossible. He believes that exploratory operation is justifiable when the symptoms are particularly severe.

Legueu also reported before the same society a case in which there was apparently a severe nephritic colic in the left kidney. No calculus was found during the nephrotomy, but the operation afforded relief.

Such cases as these, reported by men of wide experience, fully conversant with the entire symptomatology of renal disease, show how easily mistakes can be made, and demonstrate most clearly the necessity of a more accurate diagnostic method.

There are also many conditions in organs outside the kidneys that give rise to symptoms that involve a differential diagnosis from calculous disease. This is especially true of the right kidney, which lies in close proximity to the liver, the ascending colon, the appendix, and, in the female, to the ovary. A case reported by Tédenat serves as an illustration of these difficulties. He found the symptoms of pain and hemorrhage that were nephritic in their character to have been produced by the pressure upon the kidney of an intestinal cancer.

The cases of renal disease that simulate calculus or some of those conditions outside of the kidney that might be confused with it are not the only difficulties in the way of a differential diagnosis. There are cases of calculus in which there are no symptoms or in which the symptoms are referred to some other organ. It is in these cases of unsuspected calculus that the greatest danger lies for the patient. Mr. Henry Morris says of such cases: "If such silent, lurking calculi could be discovered and removed, many deaths from calculous anuria, much illness and suffering from perinephric abscesses, and renal fistulæ, and many kidneys undergoing atrophy and degeneration might be saved by well-timed operations."

Besides these there are cases of quiescent calculus—calculi that have made their presence known by a previous attack of renal colic—where all the symptoms have subsided and the patient is in apparent health, although no calculus has been passed. There is no greater error than to tell such patients that there is nothing to be done, that the stone may pass of itself, and that the danger is passed. So long as a calculus remains in the patient's kidney there

is imminent danger. Calculous anuria may set in at any moment, and be followed by death. It is impossible to tell what the condition is in the other kidney. The absolute positive or negative diagnosis should be made as soon as possible, and if a calculus is present it should be removed.

We know that there must be many cases in which impaction takes place, and after a series of attacks of nephritic colic, during which the calculus is pushed along through the ureter, it finally becomes quiescent. The intrapelvic pressure soon equalizes the intranephritic blood-pressure, the pain ceases, the kidney ceases to secrete, and finally undergoes atrophy and complete loss of function. That such cases occur without a correct diagnosis of the symptoms being made is certain from the numerous cases reported in which after anuria both kidneys have been found totally destroyed by calculous impaction, as also in the cases so frequently met with in post-mortem examinations. Many of the cases "cured" by medicinal treatment may find their explanation in this condition, and many quiescent calculi may owe their quiescence to their impaction in the ureter and the total destruction of the function of the kidney in which they originated. Thus the danger of anuria from the formation or presence of calculi in the other kidney, and their impaction, is increased, for one kidney is already destroyed. It is easy to appreciate from these considerations how dangerous it is to allow a case of calculus, no matter how quiescent or how completely the symptoms may have been relieved, to go undiagnosticated and untreated by the removal of the calculus.

We now possess the means of making such diagnoses. The presence or absence of all calculi can be fully established and their exact location in the ureter determined. Is it right to allow a patient to run the risks of the total destruction of one kidney because we believe that the cessation of symptoms means a cure? It might have been when we possessed no absolute method of diagnosis, and when operation was attended by a high mortality. We are able now to make the diagnosis, and it has been shown that exploratory nephrotomy and early nephrolithotomy have no greater mortality than litholapaxy. The indications, therefore, for operation are very evident. We do not need to wait for complete anuria,

and should strive to prevent the destruction of the first kidney attacked while the other is fully capable of carrying on its double duties.

Exploratory nephrotomy has been one of the most valuable aids in these obscure cases that we have had, and it has enabled us to gain much valuable information regarding the conditions that simulate calculous disease. The frequency with which it has been resorted to by the best diagnosticians shows how needful an absolute method of diagnosis is.

Such are some of the difficulties that surround the differential diagnosis of renal calculus. We have seen how it is imitated by other pathological processes; how calculi grow within the kidney without giving rise to the classical symptoms, and utterly destroy its functional activity before they are recognized. We find that clinical experience has demonstrated the need of early absolute diagnosis and speedy operation, and yet we find that the symptoms are not clearly enough defined in many instances to make this possible. It is at such an opportune time that the absolute positive and negative diagnosis of renal calculus by means of the Röntgen ray has been placed in our hands. Those who understood the difficulties and knew the value of an early diagnosis hoped, when Röntgen's discovery was announced, that this method might make the diagnosis of renal calculus easy.

Early experimentation demonstrated the fact that only the more opaque forms of calculi could be detected by the rays then capable of penetrating the lumbar region, as the calculi were found to vary in their relative opacity and the less opaque were readily penetrated. The positive diagnosis was thus the only one that was valuable.

During some studies of the value of the different qualities of Röntgen discharge the author found that rays given out by what is now termed a "soft" tube would produce a contrast in the shadows of the less dense tissues, such as muscular, tendinous, and adipose tissues. From these experiments it was concluded that if a sufficient volume of low vacuum discharge could be obtained it would be possible to penetrate the lumbar region and differentiate between all forms of calculi and the surrounding tissues.

The invention and development of the self-regulating X-ray tube by Mr. H. Lyman Sayen, of this city, made possible the production and maintenance of the necessary volume of discharge in a low vacuum. This would be impossible with any other than a self-regulating tube unless it was connected with an air-pump while in operation, for the vacuum is gradually raised by the discharge through the tube, so that the rays emitted later will penetrate the less opaque calculi and destroy their shadows.

This makes the technique of this method of diagnosis difficult. An exposure that is too long or with a quality of Röntgen ray that is too penetrating will destroy the detail of the lesser densities in the negative and make an imperfect diagnosis. With too short an exposure or too "soft" a tube the differentiation is not sufficient. It is, therefore, necessary to adapt the light and exposure to the individual case. As the shadows produced vary so slightly in their densities great care must be exercised in reading the negative. All, however, that is essential to the absolute positive or negative diagnosis are two or three negatives in which the detail of the softer structures is clearly defined. Where the outline of the kidney can be readily made out it is certain that no form of calculus can escape detection. We thus see that it is not only possible to detect calculi that are present, but also to demonstrate the absence of all calculi, no matter what their density. This is the step that has made this method of diagnosis of the greatest value where renal calculi are suspected.

That such definition and detail are possible, and that an absolute positive or negative diagnosis can be secured in all cases, has been proved by clinical experience. In all of the twenty-one cases which have been studied since an absolute diagnosis was found practical the outline of the kidney itself has been demonstrated in the negatives, thus making it certain that no calculus could have escaped.

Of these twenty-one cases, eleven were subsequently operated upon, with an absolute confirmation of the diagnosis in each instance. In six of the cases calculi were diagnosed, and the diagnosis was confirmed in all its details by the operation. Multiple calculi were found in three cases, while in one calculi were present in both kidneys.

The practical value of this method of diagnosis, outside of the absolute knowledge of the presence of the calculi, was very manifest in these cases. Where multiple calculi were present the detail regarding their number and relative position was very valuable, as in two instances the surgeon under other circumstances would have been satisfied with the removal of one calculus. In one case the second calculus was completely encysted, while in the other the calculi were in a deep diverticulum of the inferior calix, beyond the reach of the exploring finger when passed through the first incision, and were only found through a second incision in the cortex based on the data obtained from the skiagraph.

In another case the calculus was quiescent and almost unsuspected, the only symptoms were a slight amount of pus and albumin and an indefinite, intermittent pain in the lumbar region. The presence of a small calculus was shown by a series of skiagraphs, and the operation, based upon the Röntgen ray diagnosis, demonstrated its correctness.

In two other cases the positiveness of the diagnosis was of great value. It encouraged the operator in a persistent search that was rewarded by the removal of small calculi that might have been overlooked.

The ease in which calculi were discovered in both kidneys is of great interest. In regard to such cases Mr. Henry Morris says: "In several of the fatal cases in my list of nephrotomy and nephrectomy for stone the opposite kidney has been proved, by post-mortem examination, to be disorganized by unsuspected calculus."

The frequency with which such cases occur both in clinical work and post-mortem examinations shows how necessary a method of diagnosis is that can determine beforehand the condition, as regards calculus, of both kidneys. The very nature of lithiasis makes it highly probable that both kidneys are frequently involved. The only solution so far advanced is the exploratory incision of both kidneys. The Röntgen ray diagnosis would seem to be a most efficient means of determining this vital point and also of determining the presence or absence of impacted calculi in the ureter.

The great value of the Röntgen ray method of diagnosis is in the possibility of detecting these calculi in their incipiency. Mr.

Henry Morris has shown, by the statistics of his operations, that the advantages of an early diagnosis and operation are very great. His statistics are: nephrolithotomy (or early operation), mortality 2.9 per cent.; nephrotomy, 23.25 per cent.; nephrectomy, 29.1 per cent.

The value of early operation before the kidney has become disorganized, before its function has been partially or totally destroyed and before infection and suppuration have set in, is clearly demonstrated by these statistics. In addition, where the calculi are small, quiescent, or just suspected, the danger of impaction and consequent anuria is guarded against, while early operation before a pyelonephritis is present will avoid the dangers and annoyance of a suppurating urinary fistula, such as frequently follows late operation.

The detection of unsuspected calculi is a great field, in which the value of this method can find no better expression than Mr. Morris's words, already quoted: "If such silent, lurking calculi could be discovered and removed, many deaths from calculous anuria, much illness and suffering from perinephric abscesses and renal fistulæ, and many kidneys gradually undergoing atrophy and disorganization might be saved by well-timed operations." These calculi can be discovered.

The value of such an absolute diagnosis in cases of calculous anuria cannot be overestimated. In these cases we have the ureter or the pelvis of the kidney blocked by a small calculus, while the second kidney is absent, atrophied, or its functional activity has been previously destroyed. The condition of the patient has no bearing in these cases, the attack comes on suddenly and with severe symptoms. The entire functional activity of the kidneys has been abolished. Acute pain may be experienced from the outset, but here is one of the difficulties in the way of a diagnosis. It may only last a day or two, or it may be that no symptom of pain and no previous history of renal colic can be obtained to guide the surgeon in determining which kidney to operate upon. Pain localized in one kidney may be a misleading guide, as was shown in the case reported by M. Léonte, who operated in a case of anuria where there was marked pain and colic referred to the right kidney.

The patient died, and the autopsy showed a calculus impacted in the left ureter. In such cases the application of the Röntgen rays will show the exact location of the calculus and determine the seat of operation.

The localization of the exact point of the obstruction is one of the greatest difficulties in the way of successful operation in these cases. If the calculus is impacted in the ureter the only method of detecting it is by direct palpation either through the rectum, vagina, or the abdominal wall. These methods are very unsatisfactory, and it is usually necessary to operate upon the kidney and explore the ureter by retrograde catheterization.

The value of the precise localization is, therefore, very manifest, as the whole operation can be planned before it is commenced, and be confined to the ureter alone if the impaction is there.

The advantages of the Röntgen ray method of diagnosis in cases of renal calculus, as demonstrated by clinical experience, are its absolute accuracy, positiveness, and comprehensiveness; its freedom from pain, shock, and danger, and, in, addition all the advantages that accrue from an early absolute diagnosis. Completeness of operation is assured by the detail which it gives, while the operation is facilitated in its execution. The negative diagnosis will frequently render operative interference unnecessary and save the patient the risk and annoyance of an exploratory operation. The positive knowledge it gives regarding the presence of calculi in one or both kidneys makes operation upon the wrong kidney impossible, and gives warning of the danger when both kidneys are involved. Its value is therefore evident in all classes of cases; where there is but a suspicion it saves the patient from the menace of anuria; where the calculi are known to be present it gives their absolute location and number, and where no calculi are present it saves the patient the risks of an exploratory nephrotomy. It consequently has indications for all classes of cases, and offers a solution for the diagnostic difficulties surrounding calculous nephritis. The diagnosis it affords is absolute, both positively and negatively, and has the advantages of mathematical accuracy.

DISCUSSION.

Dr. Thomas S. K. Morton: I would not like to have this very important paper of Dr. Leonard go by without some discussion, and I certainly congratulate Dr. Leonard upon what he has accomplished in this direction. He has been a pioneer in showing what can be done with the X-rays in certain diseases of the kidney and other viscera. His series of cases, some of which I happen to know of, is exceedingly interesting. Stones have been found where they certainly would have been overlooked by the best operators had he not been at the side of the operator with his plates and insisted upon further search being made. The prints that he has gotten through these studied efforts I think are better than any I have ever seen of the viscera, and they seem to indicate that in the future this method of diagnosis will be so refined that we will be able to judge even of tissue changes in the kidneys, for by the careful study of tubes and the taking of pictures in two directions skiascopy has become, in Dr. Leonard's hands, a method of great accuracy.

My personal experience with the X-rays in renal disease is not large, but it tends to confirm the reported results. One picture was taken by Dr. Stern and the other by a resident physician. In two cases stone was suspected, and was positively shown in the negative. One of them I operated for, and found it to be a uric-acid calculus. The other proved also to be a uric-acid calculus, but it was passed under the administration of piperazine, which drug, I may say, has deprived me of a number of operations for stone in the kidney. A third case, which seemed to indicate stone, was very suspicious because of the apparently great size of the calculus which seemed to be indicated. The gentleman who took the picture said it was a soft calculus. This was in a man who had marked symptoms which might be ascribed to stones. I opened the kidney, thinking that drainage, in any event, was necessary, and found that there was simply a very great thickening of the walls of the pelvis of the kidney, but no calculus.

I have had a number of negative results, which so far have proved to be correct. I have the greatest faith now in these pictures, and think that as the method becomes somewhat specialized as a branch of science we shall not be just to our patients unless we seek out those skilled not only in the taking but in the interpretation of the prints. I have noted with satisfaction that in the early days the courts were unanimous in ruling out skiagraphs because of the great difficulties encountered in their reading. Great injustice may be done by incorrect reading of the prints by those who have not sufficient experience in their interpretation.

Dr. J. K. Mitchell: I am fortunate enough to have submitted to Dr. Leonard a case in which, had the skiagraph not been made, we should

not have discovered the fact that, instead of the one stone suspected, there were three. The skiagraph showed the difference between the two stones, which were actually in contact. Without it we should never have thought of going further after the discovery of one large stone, an inch long and very rough, which was evidently the cause of many of the symptoms. It had destroyed the kidney in its neighborhood, and the patient was passing blood and pus. The case had been examined a number of times by various physicians and surgeons in New York, Boston, and the South, and diagnosis of stone in the kidney had never been made. I hear from the patient that he is passing scarcely a trace of pus, and the kidney is almost healed from the extensive injury necessary to get out the three stones.

Dr. W. J. Taylor: I have been very much interested in the paper of Dr. Leonard, and only regret that the possiblity to make the diagnosis of stone had not been given us a few years ago. I remember a number of cases which I had the pleasure of seeing with you, Mr. President—at least three—where there was every symptom of stone that the ordinary history would give. An incision made in the kidney in each instance revealed a movable kidney without any stone. The cases made prompt recovery, and their symptoms were relieved by stitching the movable kidney in place. I remember one instance of unsuspected stone in a case of Dr. Weir Mitchell. The patient died very suddenly. I made a post-mortem, and found a stone nearly as large as the end of my thumb.

Dr. F. A. Packard: I recall in this connection a patient who came to me complaining of pain in the right loin. Examination of the urine showed albumin only in evening specimen. From the extreme tenderness, in spite of the fact that he had not passed blood, I suspected stone or possibly movable kidney. I believed the albumin in the evening urine to be due either to shaking up of the stone, or displacement of a movable kidney, or twisting of the renal vessel during walking. I therefore had an X-ray picture taken, and immediately under the point of greatest tenderness there was a distinct shadow. Fortunately, however, the man who took the picture thought it might be well to take another one, on account of a possible defect in the glass. In the second picture the shadow was not present. The next examination was made after the man had jumped off the street-car on his way to my office. I found the right kidney down in the right iliac fossa. The application of a supporting truss has relieved his symptoms, and albumin has disappeared from his urine. I simply mention this because of the confusion which may arise in the use of the X-ray pictures. I felt that the diagnosis of renal stone was confirmed by the skiagraph in this case, but the second picture showed that the shadow was simply a defect in the glass.

Dr. James Tyson: Any one who has had opportunity such as I have had of testing Dr. Leonard's work cannot fail to appreciate its importance. Twice recently I have availed myself of his skill in confirming the diag-

nosis of stone in the kidney. In one instance in which the stone was difficult to find the operation would have been discarded had not Dr. Leonard insisted the stone was there. Equally important is the negative value of the skiagraph in cases of suspected stone where none is present. I have had the pleasure of seeing with you, Mr. President, two cases of this kind in which all the evidence which could be brought to bear gave reason to believe there was stone, in consequence of which operation was advised, but no stone was found at the operation.

There is only one thing we may lose from our experience with the X-ray, and that is the benefit derived from operations which, having discovered no cause of the pain, still relieve the painful attacks. It is a well-recognized fact that operation on these painful kidneys is often followed—for a short time, at least, and sometimes for a long time—by relief. Such a case may be one which quite recently came under my care with all the symptoms of nephritic colic. It was sent to Dr. Leonard, who made a skiagraph, and, much to my surprise and disappointment, no stone was found. Of course, it is not improbable that the stone may have dropped into the bladder.

I believe the assistance derived from the X-ray in the diagnosis of stone and the power to discover bacilli in the urine in cases of tuberculosis of the urinary passages are aids of the utmost value in the diagnosis of these affections of the kidneys.

Dr. W. W. Keen: I would like to call attention to a fact to which our attention has not been called—that is, that the skiagraph shows the actual shape and size of the kidney itself. This would be of diagnostic value in cases of tumor, hydronephrosis, etc., as well as of stone. I would have welcomed its aid very much indeed, had I known Dr. Leonard's success, in a case I recently saw in consultation, which resembled wonderfully a case of appendicitis, but in which a little time allowed the stone to escape into the bladder. The possibility of ureteral calculus was considered at the time of the consultation, but the weight of opinion was rather in favor of an intestinal colic or else of atypical appendicitis. There was absolutely no blood in the urine, as was determined by the microscope. I would like to ask Dr. Leonard whether a ureteral calculus lying below the pelvic brim can readily be made out?

Dr. Leonard (closing): As yet I have had no experience except in cases where impaction in the ureter was suspected. Symptoms of pain were elicited on pressure in the iliac fossa. I made a careful examination, and repeated the exposure a number of times for the purpose of determining whether or not a calculus was present. I think it is perfectly possible to make the differential diagnosis, because the opacity of the calculus added to the opacity of the pelvic bones will produce sufficient distinction between the two sides of the pelvis to make the diagnosis perfectly certain. I lately had a skiagraph of a perinephritic abscess which had opened, but the exact limitations of which the physicians were ignorant. An emulsion

of bismuth was injected into the abscess-cavity, and gave us its outline even through the pelvic bones. I used the same method in a case of gastroptosis for Dr. Pepper two years ago. I washed out the patient's stomach and placed in it an emulsion of bismuth. The shadow of the stomach was shown by the opacity of the coating of bismuth through the pelvic bones. These instances serve to show that a lesser opacity added to a greater opacity gives us a chance for differentiation. All opacities in regard to the X-ray are entirely relative. We speak of the bones being opaque, because they make more shadow than tissue. I can make the shadow of my hand through a sheet of lead. It is simply the opacity of the bone and the tissues added to the shadow of the lead that gives me a differentiation in the plate below. So that I think it not impossible that after a time we may be able to show a differentiation in tissues through the bone as well as between less and more opaque bodies.

In connection with one other point mentioned—that of the use of these pictures in court—I think Dr. Morton's remark regarding the ignorance of the jury is very much to the point. The way to combat such ignorance and its dangers is to have expert testimony on the other side to explain all the conditions which the picture shows. The picture shows nothing but what has happened, and it is easy to demonstrate the extent of the primary injury as well as the resulting deformity. I was called in court in a case which brought this forcibly to me. The patient had, I think, been kicked on the ankle by a horse. He came to me and wanted a picture of the sound foot. I was interested in the other foot, and asked him if I might take a picture of that. I did so, and last spring I received a subpœna to appear at court. In that case I was not allowed to testify. The plantiff's lawyer was very clever in his use of the picture. He knew that the jury was ignorant in regard to such things. He did not permit me to explain the picture, but let them see it, and then allowed them to draw an ignorant conclusion. I think the testimony of experts should be had on both sides; they should interpret the meaning of the picture, so that the jury can get at the truth. The plaintiff was awarded damages of about $3500. This shows very clearly the necessity of having these pictures explained. I have seen a number of cases of perfectly good union of limbs, so far as function was concerned, where symmetry of the bones was not shown by the skiagraph. The skiagraph in suits for malpractice will show for the plaintiff the resulting deformity, but it will also show for the doctor how serious the injury was and how difficult to treat, and how impossible it was to retain the fragments in position if the patient was in the least unruly. If properly employed it will be of great value to physicians in these cases.

RESULTS OF THE EXAMINATION OF THE BLOOD OF NINETY SOLDIERS ILL WITH TYPHOID AT THE ST. AGNES HOSPITAL.

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[Read March 1, 1899.]

During the latter part of the summer and the early part of the autumn of 1898 there were admitted to the St. Agnes Hospital 144 soldiers suffering with typhoid fever. The opportunity thus given for the study of the blood in a large number of cases, and among individuals of approximately the same age, and who, it is fair to assume, were in the enjoyment of vigorous health a few months before, was most favorable, inasmuch as the results obtained under such uniform conditions might be of statistical value. The scope of this work, in which I was ably assisted by Dr. Joseph Walsh, bacteriologist of the hospital, was the study of the quantitative and qualitative changes in the red and white blood-corpuscles and their relations to each other in the various stages of the disease; the determination of the quantity of the HB and its relation to the red blood-corpuscles, and finally to the search for malarial organisms, especially in the blood of soldiers suffering from typhoid fever, who were brought from southern camps, from Santiago and Porto Rico. In selecting the cases to be studied, only such were chosen in which the diagnosis of typhoid fever was supported by all the symptoms and signs which are regarded as characteristic of the disease, including the Widal test; while, with few exceptions, which are noted in the appended table, all cases with complications which could have a tendency to influence the result of the blood-examinations were excluded. Of the cases admitted to the hospital, a large proportion

suffered from some complication ; so that the number in which an examination of the blood could be of value for purposes of comparison was of necessity somewhat limited. The clinical features of all the cases were exhaustively treated by Dr. Franklin B. Stahl[1] in a paper read before this College at its last meeting. The principal localities from which these patients came were Fernandina, Florida, Camp Alger, Camp Thomas, Santiago, Porto Rico, and Camp Meade. Many of the soldiers from Southern camps, from Santiago, and to a much less degree from Porto Rico, were markedly emaciated and extremely pale—a condition, no doubt, due to indifferent food and to unsanitary surroundings, and not alone to their attack of typhoid fever. The skin in these cases was dry and shrunken, as well as the tissues under it, so that in many instances it was only with the utmost difficulty that a sufficient quantity of blood for examination could be procured. In several instances even puncture of a small vein failed to bring enough for our purpose, so that we were compelled to content ourselves with an incomplete examination. The blood in these cases was of a peculiar watery brownish-red appearance, and could not be made to stand out in the form of a drop on the surface, but immediately spread in a thin layer over the skin, thus making it impossible to draw it up into the pipette, a difficulty which could be occasionally overcome by painting a small ring of vaseline around the puncture, thus preventing diffusion over the surface.

Classified according to weeks of illness, there were examined 7 cases during the first week, 10 cases during the second week, 15 cases during the third week, 29 cases during the fourth week, 10 cases during the fifth week, and 4 cases during the sixth week. In the remaining 15 cases a satisfactory history of the duration of the illness could not be elicited, and these cases were therefore also excluded from the estimation of averages.

The Red Blood-corpuscles. One of the most striking features of our examination was the almost uniform reduction of the number of red blood-corpuscles, so that in only 6 cases (Nos. 18, 28, 30, 32, 74, 87) did the count show 5,000,000 or more red blood-corpuscles to the cubic mm. In two of these cases the high count was, how-

[1] See Philadelphia Medical Journal, February 25, 1899.

ever, only apparent, being due to concentration in consequence of severe diarrhœa, while the others were mild cases in the first and second week of the attack. In 23 cases the count was between 4,000,000 and 5,000,000 red blood-corpuscles to the cubic millimetre; in 33 it was between 3,000,000 and 4,000,000; in 25 it was between 2,000,000 and 3,000,000, and in 3 it was between 1,000,000 and 2,000,000.

The average count for the first week (7 counts) was 4,319,714, for the second week (10 counts) it was 3,875,000, for the third week (15 counts) it was 3,206,333, for the fourth week (29 counts) 3,191,680, for the fifth week (10 counts) 3,203,700, for the sixth week (4 counts) 3,525,000, thus showing the greatest reduction in the fourth week, after which there is a gradual increase. The qualitative changes which were observed in the red blood-corpuscles were usually those commonly seen in secondary anæmias, such as poikilocytosis, absence of rouleaux formation, etc.

Hæmoglobin. The reduction of the hæmoglobin was also an almost constant feature, but kept pace with the reduction in the number of red corpuscles only within very wide limits. When the count sank to a low figure it was noted that the HB was usually not reduced in proportion, so that such cases sometimes showed a higher percentage of HB than cases having a higher count, indicating that in such cases the individual blood-corpuscles contain an unusual amount of HB. A striking exception was, however, observed in case 41, in which the count showed 1,168,000 red cells and only 17 per cent. of HB. The average amount of HB for the first week was 88.25 per cent. (7 cases), for the second week 70.9 per cent. (10 cases), for the third week 70.2 per cent. (15 cases), for the fourth week 61 per cent. (30 cases), for the fifth week 57 per cent. (8 cases), for the sixth week 72.25 per cent. (4 cases). This shows that in all the cases a gradual reduction of the HB, beginning with the first week, took place, reaching its lowest point in the fifth week, after which it again began to rise. The HB therefore continued to diminish for a while even after the red cells began to increase.

The White Blood-corpuscles. Like most other observers, a decided hypoleukocytosis was noted by us in the majority of cases. In 68 out of a total of 86 cases, or in 79.1 per cent., there was found

a reduction to less than 8000 white cells per cubic millimetre; of these 2 were between 7000 and 8000, 13 between 6000 and 7000, 17 between 5000 and 6000, 11 between 4000 and 5000, 13 between 3000 and 4000, 7 between 2000 and 3000, 5 between 1000 and 2000, while in 18 cases, or in 20.9 per cent., there was an increase in the number of white cells varying in number from 21,000 (case 32) to above 8000. The average count for the first week was 5358, for the second week 4326, for the fourth week 4400, for the third week 5574, for the fifth week 4714, and for the sixth week 3933. Of the cases showing leukocytosis 4 had complications, as follows: Bronchitis (case 5), purpura (case 18), furunculosis (case 76), and endocarditis (case 41), while one case of lobar pneumonia (52), one case of endocarditis, one of gangrene of skin (70), and one case of furunculosis (23) had hypoleukocytosis. It would be difficult to account for the increased number of white cells in the other cases, for, although we know that it is sometimes observed in apparently uncomplicated cases of typhoid fever, it nevertheless constitutes a good reason for suspecting the presence of some complication, although not apparent at the time. The differential count showed that in almost all the cases without complications the juvenile forms of lymphocytes predominated, and that this was due principally to an increase in the large form of this variety of white cell. The small lymphocytes were found to predominate in 7 cases, while polymorphonuclear leukocytes predominated in 23 cases, mostly during the first week, or when complications existed. Beginning with the first week in all uncomplicated cases, a steady decrease in the relative number of polymorphonuclear cells and an increase in the juvenile forms was observed; so that at the end of the second week and at the beginning of the third this form of cell predominated. This increase, as was observed above, concerned principally the large lymphocytes; this was found in all but 7 cases, in which the small form was more numerous. Cases with complications usually showed a majority of the adult polymorphonuclear variety, although there was frequently observed a relative increase in the juvenile forms. Eosinophiles were rarely met with.

Parasitology. In 5 cases (Nos. 26, 33, 35, 40, 59) extracorpuscular or intracorpuscular bodies, which in some instances con-

tained pigment, were observed. These bodies were spherical, irregular in outline, and, as first stated, sometimes contained pigment which, at least in the intercellular variety, was usually gathered in or near the centre of the body. In two instances sluggish amœboid motion was observed, while in the rest the bodies were quite motionless. Dr. A. O. J. Kelly, who examined 7 cases, was, however, more fortunate, having observed amœboid motion in 3 out of 7 cases examined. The red blood-corpuscles containing these bodies were usually more or less altered, in a few instances a mere shell only being left. Other evidence of chronic malaria, such as pigmentation of leukocytes, usually of the large mononuclear variety, was observed. With the Plehn-Czesinsky stain the intracellular bodies stained a deep blue, although a more lightly-stained portion could usually be made out in the interior of the body. Evidences of rosette formation or of sporulation were never observed, nor did we ever succeed in finding rings, crescents, or ovoid bodies belonging to the æstivo-autumnal variety. The question as to the nature of these bodies is an interesting one.

The malarial character of those showing amœboid motion can, of course, not safely be denied, but the nature of the others is not so clear. Intracellular bodies as well as extracellular pigments are sometimes seen in anæmia, the former staining fairly well with methylene-blue, although not so intensely, but with more uniformity than these bodies. Their peculiar staining properties, however, their pigment, the simultaneous existence of pigmented leukocytes, the amœboid motion observed in some of them, and, finally, the disorganized condition of the red blood-corpuscles in which they are contained, stamp them, I think, as organisms of malarial origin. Ewing[1] also regards similar organisms, which he saw in cinchonized cases of malaria and in some cases of typhoid fever as atypical malarial organisms, and believes them to be of greater significance than conservative blood-analysts admit. The extracellular vacuolated pigmented and nonpigmented bodies have also been observed by Ewing (*loc. cit.*). He says that "this group does not include the adult tertian parasite, which in the fresh condition usually appears free from cellular element, and is almost always demonstrable by staining, nor the larger sterile forms showing pigment in vibratory motion.

In the fresh condition the appearance of cystic extracellular bodies strongly suggests an origin from malarial parasites." These cases, however, cannot be regarded as instances of the coexistence of active malaria and typhoid fever, but only of the latter disease, occurring in malarious subjects in whom the malarial element had been suppressed by the more masterful disease.[2] It is well, however, to remember that most of the cases received at the hospital had been already pretty thoroughly cinchonized before admittance, and that this may in part account for the absence of the typical organisms in the blood.

The question of the coexistence of active malaria and typhoid fever has been the subject of lively controversy during the last half century, and one which seemed impossible of definite solution until the employment of exact laboratory methods in general and the discovery of Laveran's organism in particular. Since then the subject has received renewed attention from medical investigators, with the result that up to date 31 cases, which have been studied by modern methods, have been published. Twenty-nine of these were collected by Lyon[3] from literature, to which he adds 2 of his own. Those collected by Lyon from literature are from the following authors: one by Osler,[4] one by Da Costa,[5] five by Kinyoun,[6] three by Thompson,[7] two by Laveran,[8] and seventeen by Vincent. Of these, two of Kinyoun's and eight of Vincent's ended fatally, a mortality of 32.22 per cent. of all the cases published.

The clinical similarity between some cases of pernicious malaria and typhoid fever is so well known that it needs no further comment, a differential diagnosis being sometimes very difficult. Rose spots, hemorrhage, diarrhœa, tumefaction of the spleen, high, continued fever, sudden exacerbations, and remissions may be observed in both; nor does a positive Widal test always help us, although presumptive evidence is in favor of typhoid fever, it sometimes fails or gives a positive reaction in non-typhoid cases. The demonstration of Eberth's bacillus in the blood or stools is better evidence, but attempts to find it frequently fail, and is always somewhat difficult of differentiation. Finally the blood-count in both affections being similar cannot aid in diagnosis, so that the final determination of the occurrence of mixed typhoid and malarial infections could there-

fore not rest on clinical data alone, but required the demonstration of the malarial parasite in the blood during life, and of the typical typhoid lesions after death.

All these conditions, so necessary for the demonstration of the actual occurrence of active malarial and typhoid fever are present in Vincent's 8 fatal cases, which came to autopsy, and in which the active malarial parasite as well as the characteristic typhoid lesions were present. This observer concludes—"from the anatomico-pathologic changes which are found in the fièvre typho-palustre, and especially from the results of bacteriological and microscopical examination of the blood and viscera, we believe that we may conclude that the syndrome studied is no other than a mixed infection, due to the association of the bacillus typhosus and the pathogenic organism of paludism." An account of the fatal cases of Kinyoun could unfortunately not be found, but the importance of Vincent's observations cannot be overestimated, constituting as they do a scientific basis for that peculiar clinical picture described by competent clinicians as mixed typhoid and malarial infection. Burserius,[9] in 1781, was the first to describe such cases under the name of the proportionata; he was followed by Herman Schmidt,[10] in 1830, and by Maillot,[11] in 1836, and Felix Jacquet,[12] in 1851, in the United States, by Drake,[13] in 1854, by Dickson,[14] in 1855, and by Levick,[15] in 1863, who described them under various names. Woodward,[16] in 1863, first introduced the term typho-malarial fever, a designation which has given rise to a great deal of confusion, inasmuch as it conveyed the idea of a peculiar combination of both infectious agencies, forming a modification of both, and hence was used, especially in the southern United States, for every case of typhoid fever which presented any unusual features. Until the discovery of Laveran's parasites and the adoption of modern methods of research, the possibility of the existence of both infections in the same subject met with violent opposition. In the cases reported by Osler, Da Costa, Kinyoun, Thompson, Laveran, Vincent, and Lyon, careful examinations of the blood were made, and the active malarial organisms were found. All modern observers, however, agree as to the comparative rarity of this condition, at least in the temperate zone. Dock,[17] in Galveston, declared in 1894, that he was willing to go

anywhere, if a doctor had a case of malarial and typhoid infection, to search for malarial organisms in the blood. Thayer,[18] in his enormous experience, never saw a case, although he admits the possibility of its occurrence. Ewing (*loc. cit.*), speaking of his examinations of the blood of the soldiers at Montauk Point, says that "in spite of every painstaking effort the attempt to find a case of typhoid fever and active malaria progressing simultaneously was unsuccessful."

Ewing, however, also admits the possibility that some of the patients at Montauk suffered from this condition, but says there was no positive indication that such was the case. When typhoid fever occurs in a malarial subject, the development of the malarial organism in the blood is usually inhibited either wholly or in part, resulting either in the disappearance of the organisms from the blood until convalescence or in the presence of atypical forms, such as were observed in some of our cases. The typhoid element usually dominates the scene, but there are cases in which the malarial infection seems to be too strong to be suppressed, thus resulting in a true mixed infection.

[NOTE.—In response to an inquiry, Dr. Stahl informs me that the possibility of the occurrence of typhoid and malarial infection was constantly kept in mind, but that the sudden rises of temperature observed were due to other causes.]

No.	Age.	No. of red corpuscles in cu.mm.	No. of white corpuscles in cu.mm.	Hæmoglobin.	Kind of white corpuscles predominating.	Juvenile.	Week of illness.	Remarks.
				pr. ct.				
1	20	3,000,000	3,500	53	.....	Large	4th	
2	30	4,218,000	3,100	39	.....	"	3d	
3	22	3,220,000	4,400	70	.....	"	5th	Convalescent.
4	24	2,800,000	5,300	50	.....	"	6th	
5	22	4,250,000	3,900	68	Adult leukocytes.	.....	2d	Bronchitis.
6	25	4,270,000	4,400	96	" "	.....	1st	
7	27	3,200,000	2,500	52	.....	Large	4th	Porto Rico.
8	22	2,600,000	8,300	50	....	Small	4th	
9	35	2,500,000	?	45	.....	Large	6th	Died.
10	?	3,236,000	3,700	68	.....	"	3d	
11	17	2,520,000	8,400	40	.....	"	4th	
12	25	3,520,000	5,200	87	.....	"	4th	
13	20	4,080,000	7,500	82	.....	"	2d	
14	21	4,534,400	8,000	65	.....	"	3d	
15	20	2,800,000	4,400	65	....	"	4th	Camp Meade.
16	24	3,750,000	1,050	85	.....	"	4th	
17	26	2,000,000	6,000	58	.....	"	5th	
18	21	5,800,000	8,100	56	.....	"	?	Purpura.
19	22	2,902,000	8,100	86	.....	Small	1st	
20	24	3,840,000	1,500	70	Adult leukocytes.	.....	6th	
21	20	4,800,000	3,100	70	.....	Large	5th	Camp Meade.
22	28	3,200,000	8,300	60	.....	"	3d	
23	27	4,379,000	4,700	?	Adult leukocytes.	.....	5th	Furunculosis.
24	21	3,320,000	6,900	71	.....	Large	2d	
25	21	4,800,000	4,400	100	Adult leukocytes.	.....	1st	
26	28	3,466,000	6,900	80	.....	Large	4th	Poikilocytosis. Pigmented and nonpigmented, intra- and extracorpuscular bodies.
27	21	2,400,000	5,000	58	.....	Small	3d	Porto Rico.
28	21	3,800,000	9,500	75	Adult leukocytes.	.....	9th	Porto Rico.
29	21	2,450,000	1,900	40	....	Large	4th	Santiago.
30	22	6,400,000	3,300	70	.....	"	3d	Severe diarrhœa.
31	35	4,500,000	10,000	90	Adult leukocytes.	.....	?	
32	21	6,000,000	21,800	92	" "	.....	1st	Camp Meade.
33	34	2,470,000	11,250	70	....	Large	4th	Nonpigmented, intracorpuscular bodies showing amœboid motion.
34	21	4,180,000	5,000	90	Adult leukocytes.	.....	2d	Camp Meade.
35	18	3,100,000	8,800	60	" "	.....	2d	Intracorpuscular pigmented bodies. Porto Rico.
36	?	2,400,000	5,800	65	.....	Small	4th	Fernandina, Fla.
37	35	2,500,000	4,400	70	.....	Large	3d	
38	21	3,600,000	7,600	48	.....	"	4th	Santiago.
39	23	2,400,000	6,250	46	Adult leukocytes.	.....	9th	Santiago. Very pale.
40	21	2,575,000	5,625	62	.....	Large	4th	Pigmented intracorpuscular bodies. Porto Rico.
41	24	1,168,000	10,000	17	Polymorphonuclear leukocytes.	.....	4th	Endocarditis.
42	22	4,266,000	4,375	93	" "	.....	1st	
43	21	2,500,000	3,225	43	" "	.....	8th	Camp Meade.
44	24	1,950,000	4,400	50	" "	.....	4th	
45	23	3,400,000	6,000	62	" "	.....	8th	Convalescent. Porto Rico.
46	20	3,900,000	3,900	53	.....	Large	5th	Santiago.
47	22	3,280,000	6,200	25	....	Small	4th	Santiago.
48	28	1,800,000	?	36	.....	"	5th	Fernandina, Fla.
49	20	3,600,000	5,500	72	.....	Large	3d	Fernandina, Fla.
50	23	4,375,000	18,536	32	Polymorphonuclear leukocytes.	.....	2d	Fernandina, Fla.
51	28	4,100,000	6,450	65	.....	Large	3d	Fernandiua, Fla.
52	34	3,587,000	2,920	87	Polymorphonuclear leukocytes.	.....	3d	Complication with pneumonia, delirius. Fernandina.
53	35	4,725,000	5,000	89	.....	Large	4th	
54	28	4,100,000	4,667	82	.....	"	3d	
55	27	3,300,000	6,250	40	.....	"	7th	Convalescent.

No.	Age.	No. of red corpuscles in cu.mm.	No. of white corpuscles in cu.mm.	Hæmoglobin.	Kind of white corpuscles predominating.	Juvenile.	Week of illness.	Remarks.
				pr. ct.				
56	22	2,800,000	6,250	35	.....	Large	2d	
57	20	2,500,000	?	30	.....	"	4th	
58	34	2,132,000	6,950	40	.....	"	10th	Camp Meade.
59	25	4,400,000	8,125	88	Polymorphonuclear leukocytes.	.....	?	Pigmented and non-pigmented intra- and extracorpuscular bodies. Porto Rico.
60	25	4,950,000	5,000	54	.....	Large	6th	Convalescent.
61	26	5,800,000	5,625	100	Adult leukocytes.	.....	1st	Camp Meade.
62	?	3,530,000	5,000	71	.....	Large	?	Santiago. Pigmented and non pigmented bodies.
63	?	3,800,000	5,965	55	Adult leukocytes.	.....	4th	Endocarditis.
64	23	3,700,000	3,868	60	.....	.....	4th	
65	20	4,266,000	3,125	95	.....	.....	3d	
66	24	3,600,000	3,950	62	.....	Large	3d	Porto Rico. Had chills and fever before present attack.
67	24	3,200,000	10,625	72	Adult leukocytes.	.....	11th	Convalescent.
68	20	4,200,000	9,000	65	.....	Large	?	
69	21	3,680,000	6,489	67	.....	"	4th	Santiago.
70	23	3,400,000	2,375	75	Adult leukocytes.	.....	4th	Gangrene of skin.
71	?	3,568,000	4,000	57	.....	Small	4th	
72	19	4,875,000	2,500	60	.....	Large	2d	
73	24	4,400,000	2,500	80	.....	"	3d	Porto Rico. Nonpigmented intracorpuscular bodies.
74	27	6,245,000	5,539	92	Adult leukocytes.	.....	2d	
75	22	3,840,000	5,000	65	.....	Large	4th	
76	21	3,962,500	16,336	62	.....	"	4th	Furunculosis.
77	24	2,728,000	6,875	59	.....	"	5th	Convalescent.
78	22	3,300,000	3,437	58	Adult leukocytes.	.....	7th	
79	24	3,500,000	8,125	57	.....	Large	5th	
80	22	2,297,000	?	72	.....	"	5th	
81	24	2,000,000	4,500	73	.....	"	4th	Santiago.
82	?	2,660,000	3,125	68	.....	"	4th	
83	23	3,400,000	3,125	60	.....	"	2d	
84	20	2,200,000	1,000	60	.....	"	5th	
85	?	4,800,000	6,000	80	.....	"	3d	
86	17	2 600,000	1,666	80	.....	Small	4th	
87	19	6,225,000	2,500	95	.....	Large	4th	
88	27	2,400,000	5,000	82	Adult leukocytes.	.....	2d	
89	33	4,200,000	5,000	65	" "	.....	1st	
90	23	3,800,000	5,000	60	.....	Large	4th	

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DISCUSSION.

Dr. James Tyson: There is one point which has suggested itself to me since the reading of the papers, a month ago, in possible explanation of the rarity with which malarial organisms are found in the blood of typhoid fever cases complicated with malaria—that is, that it may be present, possibly in the juvenile form, which is much more difficult to find than in the older, more highly developed and pigmented forms. When we remember how very much more difficult it is to find the unpigmented malarial organisms it seems not impossible that, except in the hands of the most expert, the microscope will fail to reveal them, though actually present.

Dr. A. O. J. Kelly: Dr. Muehleck has referred to my having detected the malaria hæmatazoon in the blood of a few patients ill with typhoid fever at St. Agnes's Hospital. There my experience was limited. At the German Hospital, however, I was enabled to examine the blood of a considerable number of sick soldiers, the blood having been sent to the Pathological Institute without any intimation as to whether or not the case was clinically one of typhoid fever or malaria. At the last meeting of the College Dr. James C. Wilson mentioned the statistics of these examinations in connection with his report of the soldiers treated recently in the German Hospital. He stated that of 147 soldiers ill with typhoid fever, sixteen, or 10.9 per cent., gave a clinical history of directly previous malarial infection. The malarial parasite was found in thirty-five cases, 23.8 per cent. I quite agree with Dr. Tyson that the difficulty in recognizing the malarial hæmatazoon in many suspected cases is at least partly due to the fact that the majority of the parasites are the young forms. The majority of those discovered at the German Hospital were non-pigmented, intracorpuscular

bodies. There were, however, some pigmented bodies and free pigment in the blood. These observations have seemed most interesting to me, not alone because they demonstrate the occurrence of dual infection—typhoid and malaria—and justify the designation typho-malaria fever, but also because they furnish the exact laboratory proof of the correctness and reliability of the clinical diagnosis of our forefathers.

Dr. B. Franklin Stahl: I very much regret not having heard Dr. Muehleck's paper, and I am therefore not in a position to discuss the points brought out. I however saw some of the specimens in process of examination at St. Agnes's Hospital, and the thing which most forcibly impressed me was the very great diminution in the hæmoglobin. It was a reduction out of all proportion to the clinical symptoms. I simply wish to refer to it from the stand-point of a bed-side observation.

GASTROPTOSIS: REPORT OF A CASE IN WHICH A NEW OPERATION WAS UNDERTAKEN AND THE PATIENT GREATLY IMPROVED.

By ALFRED STENGEL, M.D.,

AND

HENRY D. BEYEA, M.D.

[Read April 5, 1899.]

THE following case is an instance of enteroptosis, or Glenard's disease, in which there was downward displacement of the stomach, intestines, and right kidney. The patient was a young, unmarried woman. There was no history of traumatism, nor of illness, nor of abdominal distention by pregnancy or fluid effusion, to explain the displacement of the organs. The cause, therefore, must be considered as most probably compression of the thorax by tight clothing and relaxation of the ligaments. There was not rapid emaciation. A first operation (nephrorrhaphy) was undertaken in the hope that the gastric symptoms would subside with relief of the renal displacement. There was decided improvement, but the distinctly gastric symptoms continued, and notwithstanding prolonged rest and careful diet no relief was obtained. At this time the late Dr. William Pepper, under whose care she was, and one of the writers who had immediate charge of her case, discussed the advisability of operative interference, unaware of the fact that operative treatment had been suggested and accomplished in a few cases. In this discussion the possibility of fully restoring the stomach was carefully considered, and one suggestion that presented itself, viz., attachment of the stomach to the anterior abdominal wall, was abandoned, because we were aware of the

frequency of unpleasant symptoms in cases of adhesions of the hollow viscera of the abdomen. A second suggestion—that of gastroplication in the dependent part of the organ near the pylorus —was entertained, because we felt that the symptoms of distress, etc., might be connected with strain upon this part of the muscle of the stomach. It is not unlikely that severe strain would fall upon this part, which is the most active part (in a motor sense) in health, and in gastroptosis, the most unfavorably situated, and therefore the most prone to dilatation. If, then, an infolding of this part of the organ could be effected, and thereby the burden of the motor activity transferred to a part of the organ nearer the fundus, relief might possibly be secured for a time at least. At this point in the study of the case Dr. Beyea was called in, and suggested what appeared to us a better procedure than either of those named, and the case was submitted to him for operation. The operation will be described below. The history of the case is as follows:

Mary A., aged twenty-five years, white; no occupation; home in New Jersey; was admitted April 2, 1897.

History. Father living and enjoying good health at the age of sixty-four years. Mother died at the age of fifty-eight years. Three brothers living and well, as is also one sister. One brother died at the age of thirty-eight, of unknown cause. She believes it to have been stomach trouble.

The patient had scarlet fever and whooping-cough during childhood, but since then she had enjoyed the best of health until February, 1893, which was the beginning of her present illness.

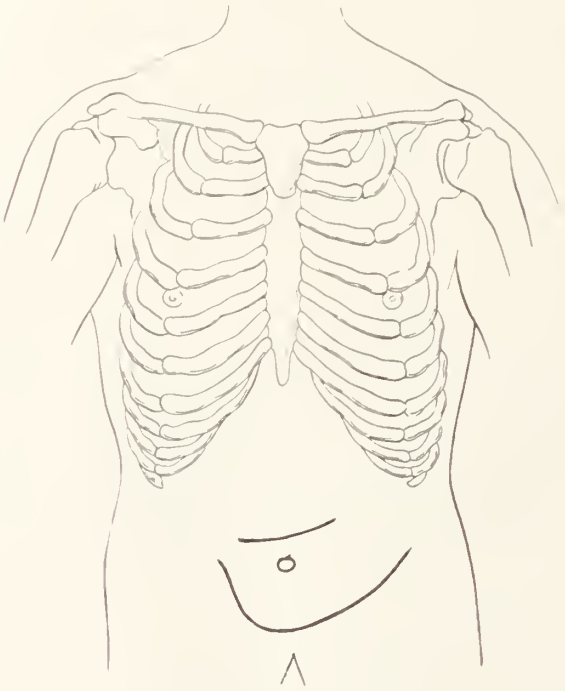
There was no history of traumatism.

In February, 1893, she noticed that immediately after eating she would have eructations of wind, lasting about two hours. The more food she ate the worse would be these eructations, and no matter what kind of food she took the eructations occurred. She occasionally had a dull pain or ache in the right flank. At present she does not complain of actual pain over the stomach, though it is at times tender on pressure. The patient has always had a good appetite. The bowels are very constipated. She has not led a sedentary life, but has always taken plenty of exercise, and she was always careful in eating, and masticated her food thoroughly.

Physical Examination. Chest ill-developed, long and narrow, especially below. The heart-sounds are normal. The lungs exhibit no abnormalities except slight "cog-wheel" breathing at the left apex. The abdomen is somewhat sunken in the epigastrium. At the umbilicus and down to

two and one-half inches below it, and extending three inches to the left and one inch to the right of the middle line, is a protuberance, soft and elastic on palpation, not tender, and giving the impression of a stomach distended with gas. There is some tenderness in the epigastrium, and just under the ribs on the right side, as well as in the region of both kidneys. The right kidney is readily palpable, dislocated somewhat downward, seems a little enlarged, is very tender on deep pressure, and is quite freely movable for an inch or more. The left kidney is not palpable. The hepatic and

FIG. 1.



Position of the stomach, April, 1897.

splenic dulness are normal. On auscultatory percussion the stomach outlines reach from one-half inch above the umbilicus (the lesser curvature) to a point two-thirds the distance from the umbilicus to the pubes (the greater curvature). The right border is an inch and a half to the right of the umbilicus; the pylorus apparently at the same distance to the right and a little above the umbilical level. (Fig. 1.) The left border is on a line with the anterior axillary fold. On distending the stomach the outlines were found to correspond with the above description. Air passes the pylorus readily. There is no palpable growth.

Three and one-half hours after a breakfast (consisting, however, of bread and coffee only) the stomach contained no remnants of the meal.

One hour after an Ewald meal, 125 c.c. of contents were obtained, containing a good deal of poorly-digested bread. Free HCl was present in slight amount. The total acidity was a little low—28. No lactic acid.

The urine and the blood were found normal.

April 8th. Six and one-half hours after a test dinner, consisting of a small Hamburg steak, two slices of bread, and 200 c.c. of water, 20 c.c. of thick contents were easily removed, after which the tube became blocked with small shreds of meat. Lavage brought out several more shreds of meat and considerable fluid contents. The undiluted contents contained free HCl in quantity, and had the very high acidity of 116. There was no Uffelmann reaction for lactic acid. Much half-digested food was present.

10th. A gynecological examination made to-day by Dr. Penrose was negative. She has a very distinct jaundice to-day. Sclerotics look very yellow. Bowels costive and stools clay-colored. Otherwise she feels the same.

12th. Jaundice persists about the same in intensity, and stools are the same. Her other symptoms are not changed.

15th. Jaundice is very much less, but still is noticeable.

During this time the urine was repeatedly examined and was practically normal, though a little too concentrated.

18th. The jaundice has disappeared.

21st. Operation (by Dr. J. William White) in his clinic; nephrorrhaphy of the right kidney. The usual incision was made in the right loin, and on exposing the kidney it was found to be quite freely movable. It was then sutured in place in the usual manner and the wound closed and dressed antiseptically without drainage.

24th. Patient complains of a great deal of pain at the site of operation. A little albumin was found in the urine in two examinations after the operation, but there were no casts.

27th. Wound dressed to-day and is entirely healthy.

28th. Some slight temperature, which is due to tonsillitis.

30th. Temperature again shows a tendency to fall, and she feels very comfortable.

Soon after this the patient left the hospital.

Physical Examination on Readmission, October 9, 1897. The general appearance is distinctly improved. She looks heavier and has a better color. The examination of the chest organs shows entirely normal physical signs. The abdomen shows the same appearance as when last in the hospital. There is a prominence from an inch above the umbilicus to within an inch and a half of the pubis, which is roughly of the shape of the stomach, is tympanitic and elastic. Percussion and auscultatory percussion indicate more strongly that this is the descended stomach, of about normal size, and inflation confirms this and shows that the location and outlines of the

organ are not changed since her discharge. The liver and spleen are of normal size, and in normal position. The left kidney seems indistinctly palpable, but not movable. The right kidney is readily palpable, and is below its normal position by one inch or more.

12*th*. Lavage four and one-half hours after administration of a test dinner of Hamburg steak, a roll, and a glass of milk showed a considerable retention of fairly well-digested meat. The stomach walls are lax, and the fluid flows in readily and is returned by siphonage very slowly. A quart and a half of water is retained at a time, and the walls can be well distended with air without causing distress.

14*th*. She shows no change in condition or symptoms. Has a good deal of burning in lower epigastrium, usually several hours after meals. She has irregular, tender joints, and complains of a "weak" and hollow feeling.

Third Admission (April 11, 1898). Her general appearance is distinctly improved. She has a better color, and has apparently gained flesh.

She complains of the same old trouble returning. Has a good deal of pain at irregular points over the abdomen, and especially some hours after meals there is a burning pain about the umbilicus, and distention with gas, and belching.

The right kidney region is often painful. The kidney may be felt somewhat below the normal position, but it is slightly if at all movable. The left kidney is not distinctly palpable.

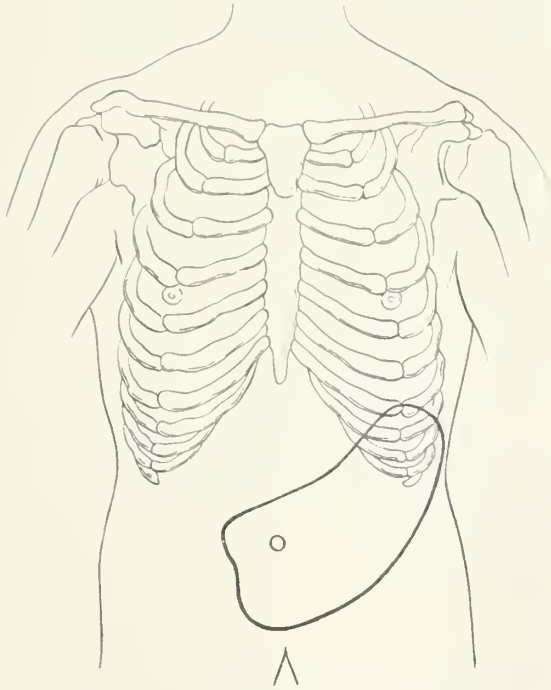
The stomach is seen, upon simple inspection, in the same position as before. Upon percussion, however, and especially upon percussion after inflation, there seems to be some increase in the size of the organ. The pylorus is at least two and one-half inches to the right of the umbilicus, and nearly on a level with it. (Fig. 2.) The portion of the stomach lying to the right of the umbilicus is distinctly enlarged, and when distended the vertical width of the stomach extends from one and one-half inches above the umbilicus to about one and one-half inches above the pubis. The fundus reaches up to the sixth rib in the anterior axillary line or a little anterior to that line.

The patient was now transferred to the gynecological wards, and the operation was performed on April 19, 1898.

Operation. The patient, after having been prepared for the operation of cœliotomy and anæsthetized, was placed upon the Boldt operating-table in a position opposite to that employed for operation through the lower portion of the abdomen. The object of this position was to permit the elevation of the chest and upper portion of the abdomen, which would allow the gravitation of the intestines and stomach out of the field of operation, and give the greatest operative space, in order to gain every advantage of the Trendelenburg position. The skin of the abdominal wall was then scrubbed with green soap and water, ether and alcohol, and the field of operation surrounded by sterilized towels. An incision about four inches in length

was made through the linea alba, midway between the xiphoid cartilage and the umbilicus. The tissues were separated in the usual manner and the peritoneal cavity opened, exposing a small portion of the upper curvature and cardiac end of the stomach, the gastro-hepatic omentum, the gastro-phrenic ligament, and the lower portion of the liver. The table was then elevated to the Trendelenburg position, and the stomach displaced still further downward and out of the wound by means of gauze sponges. This procedure caused the gastro-hepatic omentum and gastro-phrenic

FIG. 2.



Position of the stomach during the third visit to the hospital (April, 1898), just before operation.

ligament to be slightly stretched and separated from the underlying structures, which permitted an accurate determination of the length of the gastro-hepatic omentum and very much facilitated operative manipulation. The gastro-phrenic ligament was seen to be well developed, and evidently formed a strong support for the cardiac end of the stomach, while the other portion of the gastro-hepatic omentum was composed of thin, delicate peritoneum, but was determined to be of sufficient textural strength to hold

sutures, and also gave much support. Retractors were now introduced and the liver held aside by the fingers of an assistant. Interrupted sutures were next introduced to shorten the gastro-hepatic omentum in the following manner: The first suture caught the gastro-phrenic ligament above at a point as near as possible to the diaphragm (a distance of about two inches from the diaphragm) and below, just above the gastric vessels. After this suture had been placed the gauze sponges which displaced the stomach were removed and the suture temporarily tightened, so as to determine the height to which the stomach could be brought by this manner of suturing and the degree of support which would be furnished to the stomach. This having been determined to be satisfactory, the stomach was again displaced out of the wound and the suturing continued. The second suture was introduced through the gastro-hepatic omentum opposite, and about one-fourth of an inch from the first one, followed by a row of eight or ten others introduced in the same way, to include the left three-fourths of the gastro-hepatic omenum. In order to be sure of gaining a wide surface of adhesion, particularly in relation with the gastro-phrenic ligament, three or four more sutures were introduced, which included the peritoneum above and below those already placed. The gauze sponges were again removed, and the first and then the second row of sutures were tied, forming a tuck in the gastro-hepatic omentum and gastro-phrenic ligament. After this had been accomplished the stomach was seen to occupy what was thought to be a normal position. All gauze sponges were then removed from the abdominal cavity and the table lowered until the patient was in a horizontal position. The wound was closed by suturing the peritoneum with fine silk, the fascia with silver wire, and the skin with fine silk, with an intracutaneous stitch. The wound was dressed with two layers of sterile gauze held in position by antisepticised collodion, covered by several more layers of sterile gauze. In order to assist in giving support to the stomach and aid in preventing traction upon the sutures during the early part of convalescence a large compress of gauze and cotton was placed over the lower two-thirds of the abdomen, and the dressings and compress held in place by a many-tailed flannel binder.

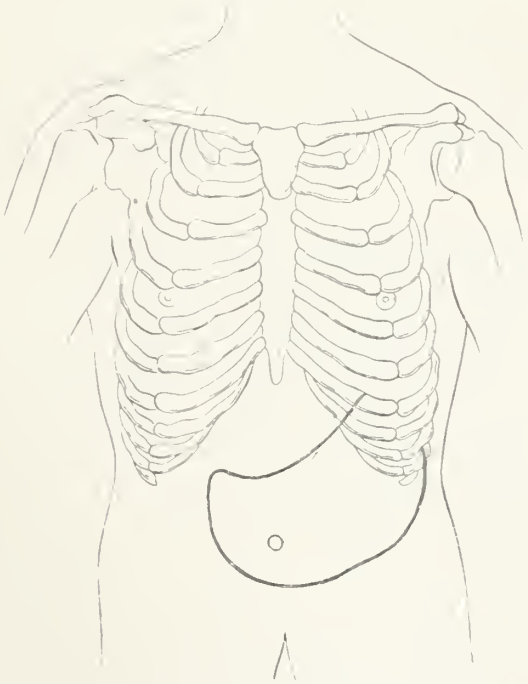
Fine silk was employed for suturing the gastro-hepatic omentum and gastro-phrenic ligament. The needles were small, curved, and round-pointed. This particular form of needle was selected to prevent tearing the tissue in the introduction of sutures. In introducing the needle care was taken to introduce the stitches evenly and accurately without causing tension. Each suture also caught the greatest possible amount of tissue practicable, in order to give the greatest strength and form a wide adhesion.

After the operation, which lasted forty minutes, the patient was returned to bed in good condition, the pulse being 98 and the temperature 97° F. She made a normal convalescence. After the first five hours the pulse was never more frequent than 78, and the temperature but once reached 100° F.

She was given nothing by the mouth for forty-eight hours except small amounts of hot water, at the end of which time drachm doses of milk and lime-water began to be administered hourly. The amount of milk was gradually increased after twelve hours, until, on the fifth day after operation, she was taking five drachms of peptonized milk every hour. The bowels were opened by an enema thirty-six hours after operation. She was kept in bed and upon her back until June 28th, and left the hospital on July 1st.

After her discharge from the hospital she wrote us from time to time about her condition. After sufficient time had elapsed we requested her to return to us for examination. The results of this examination are recorded in the next paragraph.

FIG. 3.



Position of the stomach November 10, 1898, and March 14, 1899.

November 10, 1898. The patient visited the hospital to-day, reporting a gain of ten pounds in weight, and was feeling somewhat better than previous to the operation. A stomach-tube was inserted and the stomach inflated with air. This showed that the lesser curvature was still well supported, and the greater curvature did not extend more than an inch and a

half below the umbilicus, taking the lowermost border of the sloping projection caused by the inflation as the situation of the greater curvature. (Fig. 3.)

March 14, 1899. The patient again returned to the hospital upon our request, and states now that she is very much better, and has gained nine pounds since her last visit. She is able to eat freely without discomfort, has a distinctly better appetite, and feels inclined to do more work about the house. The stomach was inflated with air and was found to occupy exactly the same position as that discovered at the last examination. Palpation of the kidney reveals the fact that it has been sustained in its normal position. Twenty-four ounces of water were then allowed to flow through the stomach-tube, when the patient complained that the stomach felt full. The tube was carefully removed, and the gastro-diaphane was inserted. When the light was turned on a bright spot of illumination was observed (umbilicus) and diffuse illumination of the area, which had been prominent upon inflation, and having almost the same superficial boundaries. The results of this examination were, upon the whole, unsatisfactory on account of the haziness of the outline of the light and the changed relations with changes in position of the patient and changes in the tube within the stomach.

We have made a careful search through medical literature to determine what operative measures have been used for the relief of this condition, and have found the cases which are abstracted below. It will be observed that in none of these was the operation exactly similar to that which we undertook, although one of the operators (Davis) describes a very similar operation for the relief of descent of the small intestine which accompanied the gastroptosis.

Treves[1] records an interesting case, as follows: The patient, a young lady, aged twenty-two years, had been ill for six years. She was highly cultivated, but had never been considered as a neurotic subject, nor did she appear so. The illness began six years before with an acute abdominal disturbance, supposed to be an ulceration of the stomach or intestine. The symptoms rather pointed to an intestinal trouble, as far as could be made out from the story of the patient. There had been a fall previous to this illness, and she never fully recovered after it. The symptoms in general were those associated with Glenard's disease. Abdominal pain was almost constant, vomiting was common, and the vomited

[1] British Medical Journal, January 4, 1896.

matter was intensely acid. She was much worse when in the upright position. A right floating kidney had been discovered and had been fixed in place by a surgeon three years before, and it still remained in good position. The rest treatment was tried, but the patient grew worse under it. Finally she had found relief by wearing a belt, but the latter had to be made increasingly stronger to support the viscera. Treves operated in June, 1895. The abdomen was opened in the middle line a little below the xiphoid cartilage. The liver was found presenting at the wound, and the stomach occupied a lower level than usually, but it was impossible to drag it upward. This was found to be due to the fact that the greater omentum was rolled up into a rigid cord, which was fixed to a mass of stony hardness in the right iliac region. A second incision was made of this mass, and it was found to be a collection of old, tuberculous glands. These were at once removed. The stomach then became movable. The liver was secured in place by ligatures. These concerned the lines of the falciform ligament and the umbilical fissure, the most important stitch being passed through the liver near its edge and penetrating the round ligament. The other stitches involved the round ligament and the falciform ligament. Above, the sutures were passed through the fibrous tissue of the abdominal walls at the side of the xiphoid cartilage. The patient made an excellent recovery without complications, excepting a small stitch abscess, and the troubles depending upon the ptosis of the viscera disappeared. She was able to walk without discomfort. The gastric symptoms almost disappeared, and five months after the operation, when the patient left England, the only complaints were of weakness and some intestinal pain which may have been due to adhesions.

Duret[1] describes a case of gastroptosis in which he performed a novel operation, which he designates "gastropexie." The patient was a woman, aged fifty-one years, who had been in good health up to a comparatively recent period. She was married at twenty-two years of age, and had two children. She had had some nervous symptoms which were attributed to cervical metritis and displace-

[1] Revue de Chirurgie, 1896, p. 421.

ment of the uterus. Three years before she came under observation she had suffered with bad digestion, due to dietary irregularities. The symptoms of gastric disturbance coincided with the first days of her periods. The dyspeptic troubles increased, and she lost weight, the decrease amounting to seventeen and one-half kilos. No improvement followed treatment of various kinds, and she came under the author's care in a much emaciated state, though her face did not show the evidence of severe disease, the skin having a rather healthy color. The abdomen was hollow, and on palpation in the iliac fossa the cæcum could be found distended with fecal matter. In the left iliac fossa he discovered a hard mass, which could be rolled under the finger, and which was the sigmoid flexure turned upon itself, small in size, not being larger than the index-finger. The transverse colon could not be plainly palpated. The liver was moderately depressed or enlarged; it extended about the width of the finger below the ribs. The spleen was not accurately delimited. In following the procedure of Glenard he could not determine any movability of the right kidney. On percussion the epigastric region was hollow, and superficial palpation determined the aortic contractions very plainly. The stomach could not be delimited by percussion, but on examining the splashing sounds it was discovered that the greater curvature extended to a point about four fingers' breadth above the pubes. Lavage of the stomach was practised, partly as a therapeutic measure and partly for diagnostic purposes, and it was found that it was possible to introduce a quantity of water, amounting to a litre and a quarter, without producing a tendency to vomiting. It was finally concluded to undertake an operation, and the author proceeded as follows:

After the abdominal incision was made from a little below the ensiform cartilage downward 8 or 10 cm., sparing the peritoneum in the upper end of the incision, sutures were inserted through the peritoneal reflection of the stomach, at the junction of the lesser curvature and anterior wall of the stomach and through the peritoneum in the upper angle of the superficial wound, and the stomach thus fastened in place. The abdominal wound was then closed. The patient's general condition improved very much after

the operation, and she was finally discharged, having begun to take ordinary food. The functions of the bowels were normal. Digestion was no longer painful. The tongue was clean, and she was able to take all sorts of food, including bread, which had been very difficult to digest before. She had gained in weight.

Byron B. Davis[1] records two cases in which operation was undertaken for the relief of gastroptosis. With regard to the diagnosis, he states that "if the lesser curvature is found halfway or more from the ensiform cartilage to the umbilicus, no doubt can exist that ptosis not only of the stomach but of the intestines is present." Some form of treatment must be devised which will support the displaced parts, and the functions of the intestines must be regulated in some way. He notes that the only surgical procedures undertaken prior to his report were gastropexy and gastrorrhaphy. Shortening the mesentery, which is the method pursued by himself, had not been attempted before, nor had shortening of the gastro-colic omentum been practised.

Case I.—A farmer, aged sixty-three years, presented himself with a large abdominal hernia, extending from two inches above the umbilicus to the symphysis. When he stood erect the recti separated for a distance of three or four inches, and a considerable part of the abdominal contents was extruded. This hernia made its appearance six or seven years before, and was caused by a laparotomy for the relief of an omental or intestinal tumor. The patient suffered with pain in the umbilical region, and great discomfort in the lower part of the abdomen when he stood erect. There was also a left inguinal hernia. An attempt to cure both herniæ was made in February, and when the patient returned in July there was no recurrence of the herniæ, but he still had great discomfort and pain in the umbilical region, and it was supposed that adhesions had re-formed. The following operation was undertaken: A median incision was made from near the ensiform cartilage to an inch below the umbilicus. The adherent omentum was detached and a part ligated and removed. Then the stomach was drawn up into its natural position, and the lesser omentum near its reflection from the stomach was fastened to the peritoneum near the ensiform with fine silk sutures. The stomach was not greatly dilated, the transverse colon was fully six inches below the greater curvature, and a tuck was taken in the gastro-colic omentum, the bloodvessels being carefully avoided by the sutures. This procedure shortened the distance between the colon

[1] Western Medical Review, October 15, 1897.

and stomach to two or three inches. The small intestines were brought forward, and the mesentery was found to be so much elongated that the loops of intestine could be raised four or five inches above the abdominal wall without great tension on the mesentery. In order to shorten the latter without interfering with its blood-supply he used a long needle with No. 4 silk, inserted the needle near the apex of the mesenteric triangles representing the mesentery between neighboring arterial branches, penetrated the mesentery, and brought the needle through in the opposite direction at the centre of the base of the triangle formed by its intestinal attachment. The suture being tied, a reef was thus secured in the mesentery, shortening it from two to three inches. The circulation was not impaired in the least. Sutures were thus used the entire length of the small intestine, not in every interarterial space, but almost as close at that. Altogether ninety-two sutures were inserted between the upper jejunum and the iliocæcal valve. The patient recovered with practically no shock from the operation, and did well. The bowels moved on the third day, and on the twentieth day the patient was out of bed, and left the hospital four weeks from the day of the operation. There was apparent improvement at the time of the report, in September, 1897.

Case II.—Mrs. P., aged thirty years, the mother of two children, had suffered for two or three years with great distress after eating, and on standing she felt a downward pressure and bulging in the lower abdomen. There was obstinate constipation, great headache, and nervousness. The liver dulness was found to extend downward one and one-half inches below the ribs. The lesser curvature of the stomach was an inch above the umbilicus. The motor power of the stomach was normal and the examination of the gastric contents showed normal conditions. The patient had been operated upon previously for the repair of the cervix and a torn perineum, but was practically unimproved. On September 4, 1897, an operation was undertaken for the relief of the gastric dislocation. The stomach was found as previously outlined. The lesser omentum near its attachment to the stomach was stitched to the peritoneum on a level with the ensiform cartilage; and at the same time, as the stomach was considerably dilated, gastrorrhaphy was performed, thus serving the double purpose of reducing the size of the stomach and raising the transverse colon. The operation required one hour, and there was no shock from it. The bowels moved on the third day, and on the ninth day the dressings were changed and the sutures removed.

Ferrari,[1] in discussing gastro-enterostomy in the treatment of ectasia and ptosis of the stomach, advocates gastro-enterostomy under certain circumstances in the latter condition.

[1] Revue de Chirurgie, 1897, vol. xvii. p. 337.

In the discussion Nicomi expresses opposing views, and advises only gastroplication.

Haunecart[1] describes a case of marked splanchnoptosis and relaxation of the abdominal walls upon which Depage operated with success. The operation was as follows: A transverse incision was made from the end of the eleventh rib to the opposite side at the same point. Two converging incisions were made from the ends of this obliquely downward to about the horizontal level of the umbilicus, where they were about 5 cm. distant from the middle line. From these points two arched incisions were made, which united in the neighborhood of the symphysis. After the skin of this part was separated, the linea alba, together with the edges of the rectus muscle and the peritoneum, were extirpated. The parts were brought together in a T-shape, and thus the abdominal wall was narrowed in a vertical as well as a transverse direction. The suspensory ligament of the liver was fastened in the upper angle of the wound, and thus the liver was fixed in its normal position.

Terrier and Hartmann[2] record a case, as follows: A woman, aged thirty-one years, had been ill for two years, her sickness having commenced by inability to take wine, the taking of the least quantity of this causing severe pain. She had intense constipation, some purgative being invariably necessary to secure a movement. When she came under observation she was cachectic in appearance, and on distention of the stomach this organ was found dilated and dislocated in a downward direction. The dilatation, as figured in their cuts, involved the pyloric end of the organ mainly, and was of the form alluded to in our discussion of dilatation secondary to dislocation. The operation, performed by Dr. Hartmann, was a combined gastrorrhaphy and gastropexy. A number of parallel sutures were fastened in the dilated pyloric end of the stomach, so as to form a plication, and the whole organ was then elevated and fastened to the abdominal walls. She was carefully fed for a time after the operation, and her general condition improved greatly. Three weeks after the operation inflation of the stomach showed it to be in the normal position. From

[1] Journal Méd. de Bruxelles, 1898, No. 8.

[2] Chirurgie de l'Estomac. Paris, 1899.

October 12th, the day previous to the operation, to January 10th she gained fourteen and one-half kilos.

DISCUSSION.

Dr. James Tyson: I have examined this patient since the operation, and have outlined the stomach in the position described by Dr. Stengel as the result of the operation. The position is very decidedly different from that which preceded the operation, being in almost the natural situation. The patient at the time I examined her appeared to be in very good health; was cheerful, and evinced great satisfaction from the results of the operation.

Dr. F. P. Henry said: I would like to ask Dr. Stengel what was the result of the blood examination, assuming one to have been made, because, as is well known, Meinert, of Dresden, attributes all cases of chlorosis to gastroptosis. I think the displacement in this case might be attributed to congenital relaxation or weakness of the supporting ligaments of the abdominal viscera.

Dr. A. Stengel: In answer to Dr. Henry's question I will say that the blood in this case was examined on several occasions, and was practically normal. As far as this observation goes there is no support of Meinert's theory.

SOME REFLECTIONS UPON CELLULAR PHYSIOLOGY AND PATHOLOGY.

By AUGUSTUS A. ESHNER, M.D.,
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[Read May 3, 1899.]

Modern research and investigation have shed much light on the intimate nature of disease processes, but there is a good deal of virgin soil yet to be tilled. Life may be looked upon as a manifestation of the activity of the cell (or of an aggregation of cells), which, if expended in a proper manner, represents the condition of health. Perversion of this activity, through influences acting from either within or without, leads to disease; while permanent inhibition (destruction) results in death. Normal cellular activity (function) implies a proper relation between waste (catabolism) and repair (anabolism), the replacement of used-up and worn-out material by fresh nutritive pabulum, together with the elimination of the products of disintegration—normal metabolic or nutritional equilibrium. Failure in either of these processes, as well as defect in quality of the nutritive pabulum, gives rise to derangement of this metabolic equilibrium, resulting in the one instance in inanition, in the other in intoxication. The latter, besides being intrinsic, may also be extrinsic. In any event, the derangement of nutrition is attended with alteration of function, which, however brought about, adds in turn to the nutritive disturbance. Cells differ among themselves in their activity and reactivity, in their affinities and antagonisms, in their susceptibilities and resistances, in accordance with qualities, properties, and attributes either derived from parent cells (heredity) or acquired through the influence of environment or other extraneous factor.

Disease may thus be viewed as primarily the expression of a perversion of nutrition, with derangement of function, and also with alteration in structure. The nutritive perversion may arise from changes taking place within the body or result from the operation of agencies introduced from without. Thus, nutritive disturbance and functional derangement may result, on the one hand, from failure in the elimination of waste products of metabolic activity generated in ordinary amount, or the retention of such products in consequence of their generation in excess (auto-intoxication); or, on the other hand, from the action of poisons that gain entrance into the body in various ways (hetero-intoxication). Disease may be caused besides by certain physical agencies of extraneous origin, such as violence, extreme heat or cold; and as a result of the invasion of micro-organisms (infection) and the unfolding of their activity (intoxication). Physical agencies give rise to disease directly by reason of their destructive effects, and indirectly by rendering favorable the conditions for infection and bacterial activity. Destruction of tissue means loss of function and deranged metabolism, and altered nutritive conditions. Bacteria act in part as foreign bodies, in part as parasites, and often in most important part as producers of toxic substances; and in each of these ways they cause modifications in cellular activity and in cellular metabolism. Derangements of nutrition may, if profound or if long maintained, give rise to structural alterations, and these in turn to organic change.

Applying the foregoing considerations to the process or complex of symptoms that we designate inflammation, we would say that an irritant, local or general, infectious, chemical, mechanical, gives rise first within the range of its activity to modification in the nutrition of cells in a larger or smaller area, and as a result of the metabolic disturbances thus generated other irritating substances are set free that induce further changes, as manifested by local or general heat, vascular constriction and dilatation, cellular multiplication, exudation of fluid, swelling, discoloration, pain, acceleration of heart-beat and of respiration, and other well-known symptoms. The results and the products of irritation by aseptic and non-toxic materials differ from those due to septic and toxic

substances, and both of these in turn from those due to infectious agents, by reason of differences in chemic affinity and activity, both in quantity and in quality, and in accordance with the localization or dissemination of the infective agents (micro-organisms). The resulting inflammatory changes in local cellular metabolism and activity gve rise to the local heat, the local vascular changes, the local increase in cells and exudation of fluid, the local discoloration, swelling and pain; while absorption into the general circulation of the products of the local metabolic abnormity gives rise to the pyrexia, and, alone, or perhaps in conjunction with this, to the acceleration of heart-beat and of respiration, the thirst, the headache, the delirium, the convulsions, the coma and the other symptoms, in accordance with the chemic affinities and activities of the metabolic products generated and the cellular area attacked. The inflammatory process is sometimes unattended with pyrexia, and the explanation of this apparent paradox must be sought in the chemical affinities and activities of the metabolic products generated, and reciprocally the reactivity of the nervous mechanism attacked. Bacteria are foreign bodies, and, finding lodgement in any tissue, give rise to independent local lesions and processes. Some of the products of cellular activity cause hyperplasia, just as certain nutritive conditions are conducive to the continued existence, activity, and multiplication of micro-organisms. Other of these products cause cellular necrosis and degeneration, just as many micro-organisms are destroyed by the products of their own vital activity and so-called self-limited diseases are in part terminated.

Going a step further and applying the considerations brought forward to our present conceptions of immunity, we would assume that in the natural, hereditary or congenital variety the cells of the body have been endowed, by transmission from the parent or parents, with a certain physiological activity, in consequence of which, through themselves directly (phagocytosis) and indirectly through the fluids by which they are surrounded (blood, lymph, serum) or substances that they generate (alexins, antilysins, antitoxins), they either repel the invasion of pathogenic bacteria, or, failing in this, they counteract the effects of the noxious products

to which these give rise. In acquired immunity, on the other hand, the modifications in cellular metabolic equilibrium that are responsible for bactericidal and antitoxic activity are induced through the agency of the chemical substances generated by the specific micro-organisms, either through the disease itself or through inoculation or vaccination or antitoxication. The cells are made by exercise under appropriate stimuli to evolve a latent function.

The nutritive, metabolic, and cellular processes that we have been considering briefly apply equally to normal and to morbid states and with especial fittingness to the disorders that we are in the habit of designating functional or idiopathic. Sleep may thus be looked upon as resulting from the action of certain metabolic products upon the higher cellular elements of the central nervous system; narcosis from perhaps others of like kind; thought from the action of other metabolic products; sensation and volition and voluntary action from the operation of still others; and so on, *ad infinitum*. We have long passed the threshold of a specific pathology; it is time that we recognize that we are moving in the domain of a specific physiology. In partial confirmation of this proposition it need only be pointed out that many of the states named, normal or abnormal, can be induced, at least in some degree, by various medicinal agents.

Derangement of the normal metabolism affecting especially the cellular elements of the nervous system may result in wakefulness, in delirium, in insanity, in neurasthenia, in hysteria, in chorea, in epilepsy, and other cognate disorders. Whether the neuron is motile or not is unimportant in this connection. It is probable that it is, as contractility is an essential attribute of all living things. The terminations of neurons are, moreover, not in absolute contact, but rather in approximation, and it is likely that no disturbance of function results from such changes in relation of end-brushes as is constantly taking place. That which is of real importance is the influence that is responsible for all the functions of the neuron, including its contractility, and that is its nutritive state. We would not say that darkness is responsible for the lower temperature of night merely because the two are associated,

when we know that it is the withdrawal of the influence of the sun to which both phenomena are due. No more should we, therefore, make the motility of the neuron an essential factor in the physiology or the pathology of the nervous system, as that is but one manifestation of the activity of the nerve-cell. Neurons must vary in size and shape and relation and chemical affinity and activity in accordance with their nutritive and metabolic condition, and it is changes in this state that are responsible for the variations in functional activity in both health and disease. Changes have been demonstrated to take place in the ganglion cells of fatigued animals and of those subjected to various infectious and toxic processes. Such changes, if long continued or of sufficient intensity, may give rise to permanent structural alterations, such as have also been found after infections and intoxications in both lower animals and men. In this way chronic degenerations arise and death results.

A quotation from a paper[1] read some years ago seems not inappropriate at this point:

"Every cell inherits from its parent cell a certain lease of life, at the end of which physiological death occurs. This period, however, may be influenced by external conditions. Premature death is pathological. We cannot recognize a pathological longevity. Similar laws apply to the human individual, the composite of a vast aggregation of cells of widely differentiated function. The life of man may be divided into three periods: that of evolution, of growth, of development; that of maturity, of perfection, and that of involution, of decline. The first begins with birth, the last ends with death.

"During the period of growth and development, tissue-change and destruction—catabolism—are more than offset by tissue-construction and repair—anabolism. Nutrition exceeds waste. Building-up is more active than tearing-down. With the activity of life comes the demand for the highest differentiation of function of which the organism is capable. This represents the perfect individual. For a time a condition of equilibrium is maintained. Sooner or later, however, the tide begins to turn. The equilibrium is disturbed. Waste exceeds repair. The cellular assimilative function becomes impaired, the constitution of the nutritive fluids defective. Tissues and organs are imperfectly furnished with the materials for their maintenance. Vascular changes occur. In a vicious circle, one bane-

[1] A. A. Eshner: "Arteriocapillary Fibrosis." Transactions of the Philadelphia County Medical Society, 1891.

ful influence reacts upon another. The entire organism is reduced to a condition of deterioration. Finally, the state of nutrition falls to so low a level as to be insufficient for the purposes of life, and functional activity comes to a standstill. This is the physiological process, as it occurs at advanced age, and constitutes the condition of senility. Occurring at an earlier period, however, the process becomes pathological, recognized clinically by signs and symptoms which may be conveniently included in the designation 'premature senility.'

"As the component parts of the organism depend for their sustenance upon the nutritive elements of the blood, so will their functional stability be governed by the quality and quantity of the circulating medium. Thus, we would be led to look to the blood as containing the excitant which induces the earliest changes. The function of the cell once impaired, the deleterious action of the blood would be augmented by the retention of matters which it was the part of the cell to remove, and the addition of products from the degeneration of the cell."

Other aspects of the results of derangement of metabolic equilibrium have been dwelt upon recently by Riesman,[1] who refers to the products generated in consequence of certain neoplastic and hyperplastic processes and the symptoms resulting therefrom. For this condition he proposes the appropriate designation "metabolic toxæmia." So-called internal secretions and their derangements are to be considered in the same connection.

A most valuable contribution to this subject from the chemical point of view has been made by Chittenden, since this paper was written, in a communication presented to the Pathological Society of Philadelphia on April 27, 1899.

[1] Internal Secretions; Metabolic Toxæmia. Philadelphia Medical Journal, February 4, 1899, p. 270.

OVARIAN MULTILOCULAR CYSTIC TUMOR, EXISTING FOR THIRTY-FIVE YEARS WITHOUT DESTROYING LIFE, THE WOMAN DYING FROM INFLUENZAL BRONCHITIS AT THE AGE OF SEVENTY-FOUR YEARS. REPEATED TAPPINGS.

By De FOREST WILLARD, M.D.,

AND

S. M. WILSON, M.D.

[Read May 3, 1899.]

The special peculiarity of this case is its illustration of what may be taken, not as the normal course of an ordinary ovarian tumor, but of an individual case pursuing an unusual but uninterrupted course. It shows the length of time which it is possible for a woman to live with the existence of such a tumor provided that no malignant element enters into its composition.

That it was not operated on early can be best explained by a retrospect of the condition of gynecological surgery thirty-five years ago. At that time Dr. Washington Atlee and his brother John L., who were then the leading advocates for the removal of such tumors, were still being denounced by their professional brethren as "dangerous men" and "murderers," and were even threatened with legal prosecution. Many Philadelphia surgeons refused to consult with Atlee or to be present at his operations, denouncing the procedure as unjustifiable and criminal. A few only of the profession were willing even to sanction the operation. The elder Pancoast and the elder Gross had not recognized its

utility; Agnew had only given it a trial in desperate cases, and then practically with a uniformly fatal result; Goodell had not yet been elected to his position in the University of Pennsylvania. Gynecology had not risen to a specialty, and hysterectomy was practically unknown; the opening of the peritoneal cavity was looked upon as an operation almost certain to be followed by fatal peritonitis, and both abroad and in this country the procedure was making only its slow but sure progress in the careful and comparatively clean operative work of Atlee, Spencer Wells, and others. Their mortality was great, but each case operated upon was one snatched from the grave. At that time Spencer Wells had only been ten years engaged in the great work which has since made his name so famous in abdominal surgery, and Keith had done his first operation only five years before. Only 122 ovariotomies had been performed in all France and but 180 in Germany, while in Austria there had been but twelve ovariotomies, and these with eleven fatal results. But twelve years previously the French Academy of Medicine had given forth the dictum that to do this operation "one must possess an American audacity."

In the United States several years later, in 1871,[1] aside from the operations of Atlee and Kimball, only about 250 ovariotomies had been performed, and the list of operators does not include the names of the then prominent surgeons—Gross, Agnew, Pancoast, Willard Parker, Sands, and others. Peasley had only done the operation twenty-eight times, Thomas twenty-seven, Emmet seventeen, and Sims twelve.

It is hardly strange, therefore, that upon first seeing this case thirty-one years ago that I, a young man just starting out in surgical work, and very anxious to secure a case of ovariotomy, should not have urged with greater success an operation which then appeared so formidable. It was, moreover, at that time the common practice to tap these tumors several times at least before attempting removal. Even fourteen years later Spencer Wells[2] advises tapping as a preliminary measure to ovariotomy, and in the same edition advocates primary tapping in many cases as a

[1] Biography of Ephraim McDowell, 1890, p. 136.
[2] Spencer Wells: Ovarian Tumors, p. 168.

procedure to be insisted upon, as the mortality of ovariotomy was not appreciably affected thereby.

With the lack of antiseptic methods at that time, even the operation of tapping was a serious one, and Kiwisch lost nine cases out of sixty-four within the first twenty-four hours after the first tapping. It is due to American ovariotomists to say that they were the first to discourage the method of primary tapping. In England the mortality of fourteen cases out of twenty-nine tappings was reported, but Peasley and others limited the operation to single cysts.

The injection of iodine following tapping had not entirely passed out of use. This iodine method in 1856 was claimed by Velpeau to be successful in one-half of well-selected cases. Boinet was so sanguine that he stated that one is almost always sure of a radical cure in monocysts. He claims that he had cured sixty-two out of sixty-seven cases. The tincture of iodine was sometimes used full strength, at others diluted, from one to fourteen ounces being injected. Some operators withdrew it, others permitted it to remain.

Tapping with permanent drainage had not entirely disappeared from practice.

As late as 1868 Millar[1] and Miller[2] reported cases being cured by internal medication :

The patient, from whom the present specimen was removed at autopsy, a married woman with several children, was, when first seen, more than thirty years ago, forty-three years of age. She had noticed the growth for some four years, and when seen in consultation with Dr. J. F. Wilson, had attained the size of a woman at full term of pregnancy. She was a spare woman, in fair degree of health, but was suffering greatly from the distention, weight, and dyspnœa. She was unwilling to accept the dangers of the operation, and was accordingly tapped, with complete relief of the symptoms for nearly two years, the fluid slowly accumulating. The operation of drawing off the fluid was repeated as required from time to time. Meanwhile several attacks of peritonitis, none of which had followed the operation of tapping, had bound the sac to the abdominal peritoneal wall, and by the time that removal of the ovary had established itself as a valuable and life-saving operation, it was evident from examination after the fluid had been removed, that intestines,

[1] Millar: Edinburg Medical Journal, 1868.
[2] Miller: New York Medical Journal, May, 1869, p. 176.

tumor walls, and wall of the abdomen had become so agglutinated that removal would be impossible; moreover, the patient experienced so much relief after her tappings that she was never ready to consent to any other form of interference. She was, therefore, tapped at intervals varying from eight to twenty months, when her discomfort became too great for endurance, and at such tappings fluid to the amount of about two or more large bucketfuls would be removed. The largest sac usually yielded ten or twelve quarts. From five to ten punctures were ordinarily made.

At the central mass, where calcareous degeneration had taken place, alternating with hardened areas of tumor, although loops of intestine had floated up and had become incorporated with the cysts, and had also become firmly united to the abdominal wall, yet at none of the operations was the bowel injured. By the use of cleanliness and care no peritonitis or inflammatory troubles ever followed the tappings, and in a few days the woman would resume her customary home work.

The fluid withdrawn was usually greenish-yellow in character, sometimes chocolate colored, and occasionally, after an attack of some debilitating illness, it would be bloody. At one time, after a severe and prolonged attack of dysentery, it seemed to have almost degenerated into pus, but at the next tapping, however, the fluid was of its usual character.

She passed safely through a number of acute diseases, and though always thin in flesh, yet was able to attend to her household duties nearly to the time of her death. At the age of seventy-four years she had an attack of influenza with bronchitis at a time when she was very largely distended with fluid (not having been tapped for eight months), and she died in February, 1899.

Autopsy. Abdomen distended to an *enormous* degree—circumference of seventy inches. Knowing that the mass was firmly adherent to the abdominal wall, the tumor was first reached from the upper portion of the abdomen. The sac wall was found to be so thoroughly incorporated with the abdominal wall that it was impossible to separate the sac from the parietal peritoneum, and dissection had to be made by removing almost the entire anterior wall through the planes of muscular tissue. The parietal peritoneum had been practically abolished. Numerous sacs were opened, discharging quarts of greenish-yellow fluid. Folds of small intestine were found incorporated with the tumor at irregular points, forming almost a portion of its mass. One large area contained a mass of pasty caseous material, such as is usually found in dermoid cysts, but there were present neither hair, nor bones, nor teeth. Plates of calcareous and almost bony formations throughout various portions of the tumor, were evident.

The tumor had sprung originally from the left ovary. The uterus was atrophied, but not involved in the mass.

Removal during life, at least for the last twenty years, would have been impossible without taking away a large area of abdominal wall, as well as intestines.

A hasty survey of the literature on the subject has yielded the following :

In the *Richmond and Louisville Medical Journal*, 1873, is reported a case of "Ovarian Tumor of Twenty-four Years' Standing, Treated by Tapping, Compress, and Bandage"

The patient was a single woman, aged forty-four years, who had suffered from an abdominal tumor for twenty-four years. For twenty years she suffered but little inconvenience, except from the excessive weight.

General health was good, but had been failing for a year. There was dyspnœa, with labored heart action, and kidney action was interfered with. Fifty pounds of straw-colored fluid was removed by tapping. The fluid reaccumulated, and she was again tapped ten months later, about the same quantity of a darker fluid being withdrawn. Six weeks later, at the time of the last report, she was in a comfortable condition.

In *Ovarian Tumors*, Atlee, Philadelphia, 1889, p. 181, is reported a dermoid cyst containing bone, in an unmarried lady, which had been tapped frequently during fifteen years. At the age of twenty-four the tumor was first discovered, which refilled slowly after tappings.

The tumor became adherent to the abdominal walls, and finally discharged continuously through one or two openings. The tumor contained hard, bone-like masses, very firmly adherent to the abdominal walls.

Patient became septic, and died from the continuous discharge of a pint or more of pus daily. At the post-mortem a large dermoid cyst was found, with very great adhesions to the uterus, intestines, and abdomen.

On page 188 of the same volume is also reported a dermoid cyst of forty-seven years' duration, which was seen only at post-mortem examination. There was no pedicle. The tumor had become twisted and separated, as the left ovary was absent, and there was no connection between the left side of the uterus and the tumor.

The tumor had remained quiescent for the thirty or forty years after she had passed her climacteric, owing to lack of nutrition, following this separation of the pedicle.

In Peasley's *Ovarian Tumors*, 1872 edition, p. 64, is the report of a case of ovarian cyst that had continued for twenty-five years, with eighty tappings, and another one in which the tumor had existed for thirty years, and another, probably a dermoid cyst, which had existed from the age of thirteen to eighty-eight. Harris has also reported a case[1] of fifty years' standing. Clay[2] gives the duration of the disease in one hundred and seventy-five cases before the period of ovariotomy, in fifteen of which the disease had existed for more than ten years.

Although Peasley states[3] that these tumors without surgical interference usually terminate fatally within four years after the discovery of the cyst, yet he cites cases of patients whose lives have been prolonged for several years by palliative tappings.

Duration, five to six years.	15 cases.
" six to seven years, ...	5 "
" seven to eight years, ...	4 "
" eight to nine years, ...	1 case.
" nine to ten years, ...	3 cases.
" more than ten years, ...	15 "

In spite of these statistics he argues that an ovarian cyst "not submitted to surgical treatment is as certainly fatal as any malignant disease, and after it has attained to a large size as rapidly so. The dermoid cyst and the oligocyst are not less certainly fatal than the monocyst, but only require a somewhat longer time to exhaust the vital force. The exceptions to this last statement are too few to be taken into account when we come to decide upon the treatment appropriate to ovarian cysts."

The interest in the specimen which we present to-night to the museum of the College consists chiefly in its duration of growth through thirty-five years and the benignity of its course under merely palliative treatment. At the present day it would of course have been removed in its early stage, and would not have been permitted to have so thoroughly incorporated itself to a large area of abdominal wall.

[1] Harris: American Journal of Obstetrics, August, 1871.
[2] Clay: Kiwesch on Diseases of the Ovaries. Appendix.
[3] Peasley: Ovarian Tumors, 1872, pp. 233 and 307.

A REPORT OF TWO CASES OF LAPAROTOMY FOR PERFORATION IN TYPHOID FEVER.

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[Read May 3, 1899.]

THE subject of operative interference in cases of perforation of the bowel in typhoid fever is of such importance that I make no apology in bringing before you this evening the report of two laparotomies performed for this condition during the past few weeks at St. Agnes' Hospital. Unfortunately, death occurred in both instances, but in view of the very fatal results in these cases if left to themselves, I feel that the attempt to benefit them was not only perfectly justifiable but imperatively demanded.

Laparotomies for intestinal perforation in typhoid fever have, of late, been very thoroughly discussed in Dr. Keen's work on "The Surgical Complications and Sequels of Typhoid Fever," by Finney, in the *Annals of Surgery*, 1897, vol. xxv. p. 233, and by Cushing, in the *Johns Hopkins Hospital Bulletin*, No. 92, for November, 1898; and I will, therefore attempt little more than to give the histories of these two additional cases and a few conclusions that seem to me worth emphasizing. In all instances where perforation has occurred the patients were already seriously ill from the disease, and we could, therefore, hardly expect a low mortality, especially where the additional element of an infective peritonitis is considered. Cushing states that in thirty autopsies held in fatal cases of typhoid fever at the Military Hospital at Fort McPherson, perforation was found to have caused death in six instances, one of these being of the appendix. This would

attribute to perforation 20 per cent. of all deaths, and, according to the latest German statistics (Gesselewitsch and Wanach, quoted by Cushing), 10 per cent. of the entire number of fatalities in typhoid fever are due to perforative peritonitis, its consequences need not be emphasized.

Cushing states that there were probably 2000 deaths from typhoid fever in the field hospitals and elsewhere during the late Spanish-American war; and if we accept these latest statistics as correct, 10 per cent., or about 200 of these deaths, were from perforation of the bowel and the resulting septic peritonitis. He stated he did not know of a single instance where operation for relief of the perforation was resorted to; but Dr. De Forest Willard, at the December meeting of the Philadelphia Academy of Surgery, reported a case of a soldier at the Presbyterian Hospital, upon whom he operated in October last. Dr. Nicholas Senn (Cushing) stated that although he saw hundreds of cases of typhoid fever during his military service, he was only called upon once to operate for perforation, and as the patient was moribund at the time, he refused to do so. Certainly very many of these cases should have been subjected to operation and an attempt made to save their lives. With Finney's statistics before us of fifty-two cases of perforation of the bowel in which operation was done, with seventeen recoveries, or 32.68 per cent., and which on revision, by eliminating all possible sources of error in diagnosis, make a total of forty-seven cases and thirteen recoveries, or 27.68 per cent.; and Dr. Keen's table of eighty-three cases and sixteen recoveries, or 19.36 per cent., we should urge the necessity for operation, especially when the known mortality without operation is about 95 per cent. Many of the reported cases of perforation of the intestine in this disease which have recovered without operation are probably due to the occurrence of a perforative appendicitis in the course of the fever, for if there be a perforation of the appendix the ulcerative process may be slow, thus giving sufficient time for the walling off by adhesive lymph from the general peritoneal cavity and the formation of a circumscribed abscess, as we sometimes see in ordinary appendicitis.

In two instances which have come under my own observation postcæcal abscesses formed and worked their way into the right

flank, where they were opened and drained, the patients making ultimately good recoveries. In one of these cases the diagnosis of typhoid fever was unmistakable; the patient was under the care of Dr. H. A. Hare, and I had the good fortune to assist Dr. Keen in the operation. In the other case, a young girl at St. Agnes' Hospital, the diagnosis was probable but not positive.

General septic peritonitis has necessarily a high mortality, and it is only by a very early recognition of the occurrence of perforation and immediate operation that we have any chance of success. Cushing lays down this rule, which is certainly well worthy of consideration, that "any abdominal symptoms occurring in the course of the fever are as urgent an indication for surgical consultation as is the appearance of pain and tenderness in the right iliac fossa under all occasions, and that only when this is fully realized will the mortality of these cases approach the low percentage reached in operations for acute perforative appendicitis or perforating gunshot-wounds of the abdomen."

I believe, to be successful, the abdomen should be opened at the earliest possible moment after the diagnosis is made, and that no delay whatever should be permitted for reaction, or, indeed, for any purpose whatever. In these cases which I report to-night the abdomen was opened within four hours in the first instance, and within two hours in the second, and in both instances a general septic peritonitis was present. The most important point in my estimation is that of diagnosis, and I hope very much to hear from the physicians present concerning it.

Can a diagnosis be made sufficiently early to warrant operation before a general septic peritonitis has developed—that is, in the preperforative stage?

Do recoveries ever take place after perforation of the bowel has occurred, without operation? Fitz remarks: "Since perforation of the intestine in typhoid fever may take place without any suggestive symptoms, and since suggestive, even so-called characteristic, symptoms may occur without any perforation having taken place, it must be admitted that recovery from such symptoms is no satisfactory evidence of recovery from perforation."

The symptoms usually given as denoting perforation are sudden

and severe abdominal pain, persisting with increased intensity (in one of my own cases there was severe pain for a week, and in the other for several days, before the perforation occurred), with nausea and vomiting, and a sudden fall in temperature; but in one of Cushing's cases the temperature was 105° F. at the time of operation, and the pulse was 170. This patient complained of severe abdominal pain and general tenderness, somewhat more marked on the right side, was restless, with pinched expression, while his color was cyanotic, the lips blue, and his extremities blue and cold.

The absence of liver dulness is not a physical sign of special value. In my first case the liver dulness was absent, as there was great tympanites, but in my second case this dulness was present. Finney mentions this absence of dulness as being noted in only five out of thirty-five cases.

Marked rigidity of the abdominal wall—a board-like condition as it is frequently described—is certainly characteristic of serious intra-abdominal inflammation, and I consider it of great importance in aiding us to decide upon operative interference.

All of these symptoms are those of beginning septic peritonitis, and show a very alarming physical condition, with the depression consequent upon a violent bacterial poisoning. The possibility of a condition of preperforative stage of ulceration undoubtedly exists, with a localized area of inflammation of the serosa, either with or without the passage through the intestinal wall of micro-organisms. This is exactly similar to the preperforative stage of appendicitis, and is indeed more serious, as the ileum is freely movable and less likely to form adhesions to other tissues and the formation of a circumscribed abscess. In my second case there was a mass of lymph almost surrounding the perforation in the ileum and firmly attached to the serous coat of the bowel, evidently an attempt, but without success, to wall off the perforation. In one of Cushing's cases this preperforative stage was recognized and the operation done, with the satisfactory result of saving life. It may be that adhesions take place between the omentum and the bowel, as has at times been reported; but this is so theoretical that it cannot be considered as a practical possibility; although, as is

shown in the specimen which Dr. Miller has brought here to-night, a perforation may occur and be closed by an omental adhesion. Fatal septic peritonitis may be present without perforation, as had been demonstrated in numerous cases; but we all know a septic peritonitis without operation and drainage is terribly fatal, and this possibility should urge us more strongly to operate in all cases where it is suspected.

Case I.—A man, aged thirty-four years, was admitted to St. Agnes' Hospital, March 3, 1899, suffering with typhoid fever. He had been sick for some time, and it was difficult to fix the exact date of the disease, but presumably it began about the 22d of February. He had been under the care of Dr. Stevens, and his condition had grown steadily worse, having had a hemorrhage of the bowel the day before; the pulse was poor and tympany increased. March 8th, about the eighteenth day of the disease, between eleven and twelve, he had all the symptoms of perforation of the bowel, with vomiting, and a drop in temperature from 102⅘° F. to normal. Dr. Stevens saw him shortly after twelve o'clock and advised that he be turned over to me for operation. I did not see him until three o'clock, and found him with a greatly distended belly, short respirations, pulse almost imperceptible, and finger-nails and lips rather blue. He had horrible pain, which was especially severe over the right side of the abdomen; his whole condition was one of profound poisoning. The only possible chance of saving his life being an abdominal section, I laid the matter before his mother and sisters, telling them I considered his chances for life without operation absolutely *nil*, while with operation there was a chance, but a very slim one, that he would recover, and that I considered it my duty to give him that one chance. They accepted the proposition, and told me to use my own judgment. All preparations were made for a rapid laparotomy. Ether was then administered, and in the course of a few minutes, after some struggling, his respirations became easier and his pulse fuller in volume, so that it could readily be counted. As soon as he was anæsthetized I rapidly opened the abdomen, and typhoid feces spurted out at least twelve inches. I quickly enlarged the incisions, pulled out the small intestine, rapidly passing down to within about ten inches of the ileocæcal valve, when I found a perforation of the gut through an ulcer which would admit the end of an ordinary lead-pencil. All around this ulcer the tissues were thickened and congested, and his condition by this time was so threatening that the ulcer was simply invaginated and the peritoneal coating closed over it with two layers of sutures of fine silk. The abdomen was then washed out with sterile salt solution, and some stitches of silkworm-gut introduced into the belly wall. Before, however, all these latter were in place, death occurred.

From the size of the opening in the gut and the mass of fecal matter in the belly it does not seem possible that an earlier operation would have had any effect, unless it had been possible to have recognized the preperforative stage, and by operation have anticipated the violent septic peritonitis. The whole time occupied in the operation from the beginning of etherization until the completion of the operation was just twenty minutes, and it must have taken at least eight minutes, if not ten, in the etherization before the abdomen was opened.

Case II.—A man, aged forty-seven years, was admitted to St. Agnes' Hospital, March 16, 1899, having been ill with typhoid fever ten days, and for several days he had had severe abdominal pain, but no hemorrhage. On March 28th, the twenty-fourth day of the disease, at 11.45 at night, the nurse discovered that his temperature had suddenly fallen from considerably over 103° F. to below normal; he complained of an intense pain in his abdomen, had vomited, and his condition was poor, but there was little evidence of shock. The resident physician saw him within fifteen minutes and diagnosed a perforation of the bowel. I saw him about twenty minutes past one, an hour and a half having therefore elapsed since the time of perforation. His temperature was then normal, his respirations rapid, pulse very rapid, but of fair volume. He had been delirious for days, and his abdomen was tympanitic, but not very much distended; there was great pain on pressure, and even a slight touch would rouse him from his stupor and cause him to complain. Liver dulness was present. The abdominal muscles were absolutely board-like. From the sudden and rapid fall of temperature, the vomiting, the great pain, and the board-like condition of the abdminal wall, I felt sure that there was an intestinal perforation. He was therefore given ether, the abdomen was rapdly opened, and as soon as the incision was made into the peritoneum gas escaped, but no fecal matter, only a thin, serous fluid. The intestines were very much distended, and popped out of the wound; they were caught, drawn outside, and a rapid search of the small intestine made down in the direction of the cæcum, where a small perforation was plainly seen in the ileum, about eight inches from the cæcum. This opening was not larger than the head of a small pin, and gas escaped, but apparently no fecal matter. There was a marked general peritonitis, and about the perforation there was evidence of the deposit of lymph. The whole peritoneal cavity was filled with colorless fluid, evidently the result of intense congestion. The small intestine was in very good condition. There was no pus, marked ulceration, or thickening, except about the point of perforation. The ulcer was invaginated, two rows of silk sutures rapidly placed in position, and the abdomen washed out with normal salt solution; drainage was introduced and the wound

closed by through-and-through silkworm sutures. His condition throughout the operation was without change, his pulse remaining good and his respirations somewhat easier after the belly was opened. He passed a very fair night, his temperature in the morning being 102° F., the pulse fair, and on the whole his condition was not materially changed by the operation. Death occurred in twenty-one hours from septic peritonitis.

I believe that cases of typhoid with pronounced abdominal symptoms should be looked upon as nearing the preperforative stage, and more especially all cases in which hemorrhage of the bowel has been noted, for without ulceration the possibilty of severe hemorrhage is difficult to understand. Osler states that death occurs in from 30 to 50 per cent. of all cases of hemorrhage of the bowel. I should much prefer to open a few abdomens without finding true perforation of the bowel if I could discover evidence of deep ulceration of the intestinal wall, as it would give an opportunity for re-enforcing the weakened wall of the gut by proper suturing, and providing free drainage for commencing general peritonitis.

The danger from operation in these cases is undoubtedly great, and when the shock is very profound may sometimes hasten by a few hours the fatal issue; but I do not believe it wise to wait for reaction, as Dr. Keen suggests, for the shock and lowered temperature are due to the large amount of septic material in the abdominal cavity and to the resultant purulent peritonitis, and not to the shock of the perforation of the bowel. The fact that the greatest number of recoveries occurred (see Dr. Keen's tables, "Surgical Complication and Sequelæ of Typhoid Fever") when the operations have been performed within the second twelve hours only carries out this contention; they are, as a rule, the cases where the perforation is small and the onset of the peritonitis slow. Our chances of success are in inverse ratio to the size of the opening in the gut, for the smaller the perforation the slower the extravasation of the contents of the bowel, and, necessarily, the milder the type of infection and degree of shock; while, on the other hand, the greater the fall of temperature the greater the degree of shock and general constitutional depression, the larger the opening, and the more overwhelming the septic infection. In these latter cases I fear our results will always be most disappointing, and it

is only by an endeavor to anticipate, to form our diagnosis in the preperforative stage, and to operate early, that we can hope for success.

There are very few of us who would wait for reaction before operating upon a case of acute perforative appendicitis with general septic peritonitis, for if we wait for reaction our patients will have passed beyond the point when they can be benefited; and, indeed, many surgeons now decline to operate at all in these cases. I admit the prognosis in all cases of septic peritonitis is very bad, but I have seen some few patients absolutely snatched from the grave by freely opening the abdomen and draining its septic contents.

Typhoid patients have a wonderful amount of vitality and stand operative interference and many surgical complications remarkably well, providing you can eliminate the overwhelming depression of a septic peritonitis.

I believe that in careful and repeated examinations of the blood in all cases of typhoid fever with severe abdominal syptoms, and indeed, if it be possible, in all cases of the disease, we will find a very valuable aid in arriving at a correct diagnosis, and it will enable us frequently to operate much earlier than we otherwise would possibly feel justified in doing. Cabot[1] and Thayer[2] have demonstrated that during the fever there is a tendency for the number of white corpuscles to diminish, and with the onset of any acute inflammatory process the number of white corpuscles is largely and suddenly increased.

Cushing has shown in two of his cases that there may even be a diminution in the number of leucocytes in the blood after the onset of peritonitis, when there is a large outpouring of leucocytes into the abdominal cavity; and, at first, this might seem to destroy this means as an aid to diagnosis. If, however, the blood be carefully examined and a marked and sudden leucocytosis demonstrated, corresponding to the increase in abdominal pain, and which again abruptly diminishes, we should at least suspect perforative and septic peritonitis.

[1] Clinical Examination of the Blood.

[2] Johns Hopkins Hospital Reports, vol. iv. No. 1, p. 83.

I would here quote Finney's conclusions as thoroughly expressing my own belief: First, of all the so-called diagnostic signs of perforating typhoid ulcer, most reliance is to be placed upon the development of an attack of severe, continued abdominal pain, coupled with nausea and vomiting, and at the same time a marked increase in the number of white blood corpuscles; second, the surgical is the only rational treatment of perforating typhoid ulcer; third, there is no contraindication to the operation, surgically speaking, save a moribund condition of the patient.

I would therefore urge most strongly that all cases of even suspected perforation with great abdominal pain, if accompanied by a marked increase in the number of white blood-corpuscles, be subjected to an abdominal section, that a rapid search be made for a perforation in the last two feet of the ileum, the appendix inspected, as well as the cæcum, and if no opening be discovered an examination made of the sigmoid flexure of the colon. Any especially inflamed spots should be covered in by stitching the sound serous coating over them.

Cushing has shown how necessary this is in the report of his case, where the abdomen was opened three times, first for perforation, second for strangulation by a band of adhesions, and third for a second perforation of the bowel and fecal extravasation.

Unquestionably a number of cases will be operated upon, if this plan is pursued, without any perforation being discovered; but if drainage be practised we will of necessity save many lives which would otherwise be sacrificed.

The vast majority of cases of suspected perforation submitted to operation where no opening in the gut has been found have recovered promptly from the surgical interference, and in some instances at least with apparent beneficial effect upon the course of the disease.

Welch, of Johns Hopkins, has shown that it is quite possible for micro-organisms to pass under certain conditions through the inflamed wall of the gut without there being any true perforation, and bearing this in mind we should always drain the abdomen.

I shall not go into the question of the technique of the operation, for with this we are all familiar in our work on the appendix, but

to insist upon the necessity for the utmost speed consistent with thorough work. The incision should be made to the right of the median line, as by far the greatest number of perforations are found in the lower end of the ileum, and, if there be need, a second opening made on the other side of the abdomen, if free drainage cannot otherwise be secured.

DISCUSSION.

Dr. H. A. Hare: One point in Dr. Taylor's paper which particularly interested me was in regard to the characteristic signs of perforation. I am a little surprised that he enunciates the view that the most characteristic and typical sign is severe pain. Within the last few years there have been a great many cases reported, particularly by the school of French surgeons, in which pain has been entirely absent. In other cases in which physicians have relied upon the presence of severe pain autopsy has revealed the fact that perforation had not occurred. There are so many of these reported in France and England and a few in this country that I do not think the absence of sudden severe pain indicates that perforation has not occurred. I have had myself this winter at least two cases of typhoid fever in which the symptoms of perforation began very insidiously; in which the abdomen became enormously distended with gas, and all the physical signs which we usually suppose to accompany perforation of the bowels were present, and yet the patient manifested no pain whatever. This may have been because they were so far advanced and so stuporous that only agonizing pain could cause them to give the slightest manifestation of suffering, or may have been due to the fact that the perforation was very small, and by a gradual leakage general septic peritonitis developed. This, I believe to be perfectly possible where we have more or less chronic inflammation of Peyer's patches. We can readily understand how, a small perforation occurring, septic matter escapes into the peritoneal cavity and the patient dies from perforation, although acute symptoms may not be present.

One symptom of value is the loss of hepatic dulness due to the distention of the belly with gas. In one case in which there was this loss of hepatic dulness it was found that the colon had gotten over the edge of the liver and given resonance where we would expect to find hepatic dulness.

I do not wish to say that I think severe pain in the bowel is not a sign, but I do think a good many cases occur in which this symptom of severe pain is absent.

Dr. G. G. Davis: My attention was called to perhaps six or seven cases of suspected perforation while on duty recently at the Episcopal Hospital. The first case that I saw presented unmistakable signs of perforation. In addition to the usual sign there was a distinct dulness in the right iliac region, which showed that there was localized trouble. In that case the diagnosis was easy, but the patient was in a state of collapse, and we felt sure that if any operation were undertaken she would die on the table. Therefore, the case was not touched. Among the other cases was that reported by Dr. Miller to the County Medical Society, the specimens of which he has brought before you to-night. I saw that patient in the first attack, when she had the symptoms generally regarded as characterizing the preperforative stage, with pain, distention of the abdomen, and some vomiting, but no localized symptom. I decided to wait, because the symptoms were hardly sufficiently marked to justify operation. The patient recovered from that attack. Then followed three or four other cases in which practically the same trend of symptoms was present. There was only one of these with symptoms pointing to the right iliac fossa, and in that there was a slight dulness on change of the position of the patient. In none of these cases except the last was operation advised or undertaken. Two of these cases recovered, but three or four died. This led us to believe we were pursuing a too conservative course. The general history of the cases was that the symptoms would be moderately marked, but not sufficiently to cause one to diagnose with positiveness a perforation, and by the time the patient was seen next he was in a state of collapse. In one or two the post-mortem revealed perforation, and I have no doubt but that some of the others died of perforation. The last case, that of a man, in fairly good condition, was seized suddenly with pain sufficiently severe to demand the use of morphine for its control. There was tympanitis, rise of temperature, no vomiting, but some rigidity of the abdomen, together with a dulness in the right flank, which changed on the change of the position of the patient, showing that there was probably effusion in the peritoneal cavity. When effusion to that degree occurs I think it may be accepted as evidence of peritonitis. We then decided to operate. The pulse was 140, although it was moderately strong, and the temperature 104°. Incision was made on the right side and a careful search made for perforation. The Peyer's patches were found to be inflamed and thickened, the intestines injected, and serum in the abdomen, showing an incipient peritonitis. No perforation was found. The abdomen was washed out well with hot salt solution and the wound closed. The temperature fell from 104° to 102° and the pulse from 140 to 120. The condition improved immediately and he went on to a satisfactory recovery.

As far as my experience goes perforation is difficult to diagnose in time to make it advisable to operate. I think if we wait until our diagnosis is positive, in a very large proportion of cases of collapse will be so marked that

death will follow operative procedure and very likely on the table. The question is whether we shall expose the patients who exhibit all the symptoms of this preperforative stage to the dangers of abdominal section and risk the possibility of finding no perforation, or whether we shall wait until the symptoms become marked and the diagnosis positive, when, as far as my experience goes, the bulk of the cases will be utterly hopeless.

Dr. D. J. Milton Miller: The case, of which I have here the specimens, shows how a perforation is sometimes healed. The patient, aged eleven years, had a severe attack of fever, with frequent hemorrhages. On the morning of the twenty-sixth day she complained of severe pain in the abdomen, and when I saw her a few hours later the abdomen was distended, with some rigidity and tenderness on pressure. After a plunge bath the temperature fell $3\frac{3}{4}°$. The next day the distention was much more marked and there was great tenderness. She had not vomited. Her liver dulness was diminished about one and one-half inches, and the temperature was 102°; pulse, 140. Dr. Davis saw her then, but did not think her condition grave enough to warrant operation, so we decided to wait until the next morning. The next morning she was much better. There was much less tympany, and she did not complain of any pain. She then entered upon a favorable convalescence. On the forty-sixth day, during a relapse, she began again to complain of abdominal pain. When I saw her there were tympany, tenderness, and some slight muscular spasm. The pulse was 124 and the temperature 103°. The next day the symptoms were more marked; the abdominal tenderness was quite severe, particularly just to the left and below the umbilicus. There was no change in liver dulness, and the pulse and temperature were the same. Dr. Davis saw her again, and thought she was, as he expressed it, "on the ragged edge" of a perforation. That afternoon her temperature fell 3°. She vomited all night and went into collapse, showing the characteristic symptoms of a general septic peritonitis, and dying about noon of the next day. At the autopsy two perforations were found—one closed by a strand of adherent omentum, and which had evidently occurred some time before death; the other a recent one, permitting free extravasation of intestinal contents.

Dr. Willard: The patient to whom Dr. Taylor alluded in his paper, and upon whom I operated for typhoid perforation last fall, had on the previous day a hemorrhage from the bowels, while under the care of Dr. Musser. About two hours previous to my seeing him he had been seized with violent pain in the right abdominal region, accompanied by vomiting, with considerable rigidity and absence of liver dulness. The only question of diagnosis was between another hemorrhage and perforation. Great abdominal pain and distention were present, and there had been a decided drop in temperature, with marked failure in heart force. As two hours had already elapsed and he had had a stool without blood, the diagnosis of perforation was quite positive. Within the half hour of preparation for

the operation his pulse failed so seriously that he seemed to have lost 50 per cent. of vitality, and he was in so serious a condition that I felt that he would probably die on the table. As there was no hope otherwise, however, I deemed it better to operate. Incision was made in the right linea semilunaris as the quickest route to the perforation. I was fortunate enough within one or two minutes to find the perforation, which was small and about eight inches above the ileo-cæcal valve. Many ulcers were seen, but no other perforations. There was no time for excision, but the perforative ulcer was speedily invested with a double row of silk Lembert sutures and the abdomen flushed with hot water. The operation was quickly done, but the man was in such a desperate condition that he died very speedily.

I do not think the question of speedily impending death ought to deter us from an effort to save these desperate cases. If there is actual perforation, it will be the perforation which will kill. If there is no perforation, the probabilities are that by drainage and washing out of the cavity the patient will be saved; the operation will probably not turn the scale. Of course, we cannot expect to save any large proportion of cases, for they are all in desperate condition when the operation is undertaken. I feel that the operation is a justifiable one, even though it does give an exceedingly high rate of mortality. It will not be long before we shall lower it, so as to give satisfactory statistics; the word satisfactory, of course, is used with a due realization of all the circumstances.

Dr. Richard H. Harte: As Dr. Taylor states, the mortality of this operation is not very low. In the first place, the cases come into the hands of the physician first, and in a large percentage of the cases considerable time elapses before the diagnosis of perforation has been made, and our results as surgeons are bound to be bad if these cases are allowed to wait any length of time before being operated on. If we wait until the diagnosis has been verified by pain, tympany, etc., and the patient is in collapse, operation is bound to give unfavorable results. I do not think that the operation during typhoid fever is as serious an operation as many suppose. I have only operated on two cases of perforation, but with unfavorable results. These cases usually come to us at the end of a long disease which has sapped the patient's vitality. I have opened the abdomen in two other cases of typhoid fever for other conditions, and apparently the operation did not affect the condition of the patients. I think it is better to err on the side of operation than to allow the patients to go on until perforation is made positive by collapse.

Dr. Daniel Longaker: I have listened to the discussion with a great deal of interest, and have noticed the association of the symptom of hemorrhage with the perforation in the first case reported by Dr. Taylor. I, myself, have seen one case of perforation following hemorrhage. A noteworthy point in this case was that the man was receiving plunge baths. The man had also the accepted plan of treatment—that is, he had received large

doses of quinine in conjunction with gallic acid. I think this association of hemorrhage with perforation would be an interesting fact to be brought out, and also the use of opium in connection with the perforation of the bowel. I am opposed to the use of opium in typhoid fever. I think it always increases the tympany. Another fact which is clear is that cases that have hemorrhage have deep ulceration, and cases which have hemorrhage early probably have very deep ulceration. I am much interested in this subject, because at this time I have a man of thirty-two years of age under my observation. He has always been in robust health, and at the end of the thirteenth day he had a very alarming hemorrhage. During the eighteen hours in which he has passed blood certainly twenty-four ounces were lost. This man has received no opium, in the fear of the possibility of the further complication of perforation. The question of the use of opium in hemorrhage and the possible influence of this drug in promoting the occurrence of perforation is a very interesting and important one.

Dr. Miller: With reference to Dr. Davis's statement that in one or two of his cases death would have occurred if the operation had been performed, and, probably, upon the table, I think this is true, if the operation had been done when the patients presented the symptoms of general septic peritonitis. I cannot help thinking, however, that, if we had appreciated the symptoms that preceded that condition, perhaps some of the cases would not have died. In the case which I reported this evening, for three days the patient had marked abdominal symptoms, particularly pain and tenderness, symptoms which, I take it, constitute the preperforative stage. I believe when there is recognized the existence of such a stage it is possible that more of these cases may be saved. We do not have the classical fall of temperature until the wound has taken place which allows the pouring out of the fecal contents into the abdominal cavity.

Dr. Cushing describes this preperforative stage as a slight inflammation of the serosa, due to the near approach of the ulcer to the peritoneal surface, and the symptoms which indicate such a condition are, particularly, complaint on the patient's part of pain. Patients do not usually complain of continuous pain in the abdomen, in my experience, during typhoid fever —*i. e.*, pain continuing for several hours. I believe that in some of these cases presenting so-called preperforative symptoms perforation has actually taken place, limited by adhesions, and then follow extravasation and the symptoms of collapse. One boy I saw this winter, who for three days was continuously crying with pain, and who had some rigidity with very little distention and no alteration of temperature, suddenly developed peritonitis and collapse. Another case for six days had continuous pain and tenderness in the neighborhood of the epigastrium, was then taken home, and died the next day with symptoms of general peritonitis. If there is, then, the preperforative stage sometimes present, it should put us on our guard to enjoin greater quiet of the patient and greater watchfulness. If these cases

are seen very often, every three or four hours, the correct time can often be determined upon when operation may be successfully performed.

Dr. H. A. Hare: I have been much interested in hearing the remarks in reference to diagnosing the preperforative stage of typhoid fever. I do not want to seem to be rude, but I would be glad to take a post-graduate course with any one who can teach me this condition from symptoms alone. I have enough difficulty in diagnosing the perforative stage. It has occurred to me that in Fitz's statistics he had fifty-six cases in which there was pain, fifteen in which pain was very slight, and five with no pain at all; and a number of other cases are recorded, in one of which the man left his bed by the ninth day, suffered no pain, and shortly after died of perforation.

The other point is as to when the surgeons wish us as physicians to call them in so-called perforation of typhoid fever. If the surgeons will tell us what symptoms should cause us to call them in I shall be glad to hear them. If they will tell us when they think operation should be done I shall be glad. We find, on the one hand, that Dr. Cushing makes the assertion that no case is too grave to be operated upon, and that operation ought to be performed the moment diagnosis is made. Dr. Keen says that operation should not be performed after perforation has occurred until the patient has had sufficient time to rally from the shock. In the records of the St. Thomas Hospital there is a report of a case which had all the symptoms of perforation. Abdominal section was done, and absolutely no perforation found, and recovery occurred. Under these circumstances it seems to me that to even intimate that the diagnosis of perforation of the bowel in typhoid fever is easy is intimating far too much. And, furthermore, I would like to add concerning the fact that pain is very frequently absent, that these same physicians who have noticed the absence of pain with perforation have also called attention to the fact that in these cases we frequently have a rise of temperature instead of a fall, such as we are accustomed to regard as being one of the characteristic signs of perforation in typhoid fever. Leucocytosis shows peritonitis or any acute inflammation, but not perforation necessarily.

Dr. Taylor: I have been much gratified at the discussion elicited. I agree with Dr. Hare, that it is a very difficult thing to make a diagnosis of early perforation. I came here as a surgeon, with very little experience with cases of typhoid fever, and as I stated in my paper, I asked a question of the medical men. If this diagnosis could be made, then we would accomplish our ends by early operation. As to the question of the preperforative stage, if Dr. Hare will read Dr. Cushing's paper in the *Johns Hopkins' Bulletin* he will find it laid out there at a pretty considerable length. The fact that a number of cases have been operated upon for suspected perforation and no perforation found, and that nearly all of those cases that have been so operated on in mistake for perforation, have recovered, I think goes to show very conclusively that we must take the risk in all cases

where there is a possibility or probability of a perforation at the time or of a perforation coming on, and opening the abdomen. Dr. Welch has shown that we may have septic peritonitis by migration of micro-organisms without perforation, and if we can eliminate this cause as well, I believe we can have a much larger number of recoveries in perforation than is shown by our present statistics.

And that would also answer to a certain extent Dr. Davis's remarks about his case getting well without finding any perforation.

In Dr. Miller's case, when pain persisted for such a length of time, I think it not unlikely that there was an acute infection of the gall-bladder. And this would be another reason why we should make an exploratory abdominal section, if we find the conditions warrant it. I do not mean to say that every case of typhoid fever with distended bowel and a certain amount of pain should have a certain surgical operation, but I do say in answer to Dr. Hare, that Dr. Cushing's ideas are very valuable and should receive most careful consideration.

THE PATHOGENESIS OF APPENDICITIS.

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During the fourteen months prior to January 1, 1899, it was my fortune to receive for purposes of examination four hundred and sixty vermiform appendices. These constituted all the appendices, with one or two exceptions, that had been removed by operation by Dr. John B. Deaver at the German Hospital during 1897 and 1898. The routine examination to which in general each appendix was subjected consisted in carefully weighing it, photographing it, measuring its length, its exterior diameter, the thickness of its wall, and the diameter of its lumen throughout its exent; noting its macroscopic appearances; hardening and embedding it, cutting and staining sections from at least three regions of each appendix, and studying their microscopical appearances; and bacteriological investigations of the contents of its lumen, of the exudate on its peritoneal surface, of free pus in the peritoneal cavity, and of drainage fluid subsequent to the operation. In addition to these four hundred and sixty appendices there were in the laboratory a considerable number of other appendices that had been removed prior to January 1, 1897. These were accorded a more or less cursory examination. Examinations were also made of about fifty appendices that, from time to time, had been removed at necropsy. These latter were presumably normal, and were included within the scope of these investigations for the purpose of thoroughly familiarizing myself with the appearances of presumably normal appendices, and for purposes of comparison, more particularly with

reference to minor pathological alterations in presumably diseased appendices. These studies were undertaken with the idea that a careful and detailed examination of a considerable number of diseased appendices might lead to the detection of some new features in regard to the pathology or pathogenesis of appendicitis, or that some disputed etiological factors might be elucidated or at least find some confirmation. It was thought also that the coincident examination of a few presumbably normal appendices might assist in the consummation of these objects. For various reasons it has been considered wise to limit the scope of this paper to a discussion of some features in connection with the pathogenesis of appendicitis, reserving for a future communication the detailed statistics upon which the opinions to be narrated are founded.

At the outset it may be stated that, in consonance with clinical observation, pathologically two forms of inflammation of the vermiform appendix may be distinguished—an acute and a chronic appendicitis. The inflammation may commence either acutely or chronically. If the former, the acute manifestations may subside after a greater or less interval of time, and the pathological alterations may persist as a chronic inflammation. It is thought also that if the acute manifestations be very mild they may possibly subside entirely and the appendix return to a condition indistinguishable, upon histological examination, from the normal. It is believed, however, that for reasons that will be detailed later this is of very rare occurrence. Chronic appendicitis may be the residual indications of a previous acute inflammation, or the condition may be inaugurated as a chronic inflammation. Of the acute and chronic forms of appendicitis several varieties may be distinguished, and for purposes of pathological study it is thought advisable to adopt some rational classification. The following classification, based upon the results of this investigation, but which does not differ essentially from several that have already been proposed, is suggested:

Acute appendicitis.

1. Catarrhal.
   - (*a*) Simple.
   - (*b*) Purulent.
   - (*c*) Hemorrhagic.

2. Interstitial.
3. Ulcerative.
   (*a*) Non-perforative.
   (*b*) Perforative.
4. Gangrenous.

Chronic appendicitis.

1. Catarrhal.
2. Interstitial.
3. Obliterating.

This classification is not in contravention of the clinical course of the disease. It must, however, be candidly admitted that we are not always able to distinguish clinically the different pathological varieties of appendicitis. In other words, these different pathological varieties may present analogous clinical manifestations. Again, the severity of the clinical manifestations of an individual attack frequently bears no proportion to the seriousness of the lesion of the appendix; but these facts constitute no valid reason against the adoption of a rational pathological classification. Rather do they indicate the similarity in the reaction of the body to diverse morbid conditions and the limitations of our diagnostic abilities.

The narrating of the pathological characteristics of the several varieties of acute and chronic appendicitis is also deferred to the future communication above alluded to. Directing attention, for the present, to the pathogenesis of the affection, it may be stated that inflammation of the vermiform appendix is, in many respects, such a unique disease; it differs so materially from inflammatory affections of other portions of the gastro-intestinal tract; it is often so sudden in its onset, so alarming in its aspects, and so disastrous in its consequences, that it behooves us to look for some cause or causes resident in or about the appendix itself to account for the much greater frequency of inflammation of this portion as compared with other portions of the gastro-intestinal tract, and for the preponderating rôle such inflammation plays in the etiology of peritonitis. As a matter of fact, there pertain to the appendix certain important anatomical and physiological peculiarities that must of necessity exert considerable influence in the production of diseased conditions

of that organ—that act, as it were, to predisposing factors. The most important of these are: 1. The shape of the mesoappendix. 2. The excessive length as compared with the width of the appendix. 3. Gerlach's valve. 4. The histological structure of the organ. 5. The blood-supply. 6. The nerve-supply. 7. The evidences of involution of the organ.

The mesoappendix is of importance for several reasons. It not only often acts as a predisposing factor in the causation of inflammation of the appendix, but it has also important bearings with reference to the possible results of such inflammation. The relations of the appendix to the peritoneum and *vice versa* are various. As a rule though the appendix is completely enveloped by a fold of the peritoneum and lies free within the perioneal cavity—it is an intraperitoneal organ. Exceptionally, however, its posterior surface is unprovided with a peritoneal covering. Under such circumstances the organ is in direct association with the retroperitoneal connective tissue, and this relation is of importance as influencing the course of possible peri-appendicular suppuration. Commonly, however, as already stated, it lies free in the peritoneal cavity, and is almost invariably provided with a mesoappendix. The latter is usually triangular in shape, though it varies considerably in size, thickness, and the extent to which it is attached to the appendix. In the majority of instances the mesoappendix extends the entire length of the appendix. Exceptionally, however, the tip of the organ may be free, rarely more of the organ, the mesoappendix under these circumstances being attached to but the proximal one-third or two-thirds of its length. It is, however, the size of the mesoappendix that has an important etiological relationship to inflammation of the appendix. If it have a breadth commensurate with its length, or if it be very short or entirely absent, as far as the mesoappendix is concerned the appendix will be quite straight. If, however, it be relatively narrow as compared with its length the appendix will be correspondingly curved or distorted. To a like extent, under such circumstances, the free drainage of the organ, so essential to its well being, will be compromised. As will be detailed later, when the thorough

drainage of the appendix is interfered with one of the most important etiological factors of appendicitis becomes operative.

The excessive length of the appendix as compared with its width, more particularly the calibre of its lumen, is another important etiological factor in appendicitis, and this also because of the interference thereby engendered to thorough drainage. Appendices vary considerably in length, some being very short, others very long. The average length is about 8 cm. The average diameter of the lumen, on the other hand, is but 3 mm. to 5 mm., and the calibre of the lumen often varies much in different portions of the same appendix, particularly if the latter has been the seat of previous inflammatory disease. The relation, then, of the diameter of the lumen to the length of the appendix is about 1 to 16 or 25—an evident disproportion to which must be ascribed considerable pathogenetic significance, especially if for any reason the lumen be diminished in calibre.

The exact significance to be attached to Gerlach's valve is indeterminate, and this largely for the reason that the structure itself is very indeterminate. It is usually evident, when the cæcum is viewed from within, as a small prominence of the mucous membrane, surrounding either completely or in part the orifice of the appendix. Upon close inspection it is seen to be made up of a reduplication of the mucous membrane, and microscopical examination reveals in addition to this some lymphoid tissue. The valve is admittedly an inconstant structure, but it can nevertheless be readily surmised that when present it serves to retard the entrance into the appendix of intestinal contents, and to hinder possibly to a less degree the escape into the cæcum of appendicular contents. It is this latter function that is the more important in this connection.

The histological construction of the appendix has important bearings upon the pathogenesis of inflammation of the organ. It is quite correct to state that in general the appendix conforms in histological architecture to the structure of the large intestine, but it is, nevertheless, very unwise to lose sight of the many particulars in which it presents deviations from that type. That which is

particularly the most important is the presence in the mucous membrane of the appendix of a considerable amount of lymphoid tissue. These lymphoid cells at times infiltrate, without definite arrangement, the mucosa; at other times they are collected into definite areas and form circumscribed masses that are spoken of as lymphoid follicles. It is because of this richness in lymphoid tissue that the appendix has been aptly compared to the tonsil. The amount of lymphoid tissue in the appendix seems to bear some relation to the age of the individual, and this may not be without significance in the production of inflammation of the organ. My own investigations, which have been so largely with diseased appendices, hardly warrant me in formulating a positive opinion on this subject; they have, however, led me to believe with Ribbert and Kelynack, that lymphoid elements are more abundant in the appendix during childhood than during later life. Ribbert states that the typical arrangement of the lymphoid elements into follicles is preserved until about the thirtieth year, when the follicles commence to undergo some atrophy and become more widely separated. Exceptionally, however, this physiological atrophic process may be installed as early as the twentieth year. Ribbert and Kelynack nevertheless, state that lymphoid tissue may be found in the appendices of the aged. Ribbert mentions also that in the rabbit, more so than in man, the appendix is characterized histologically by the presence of such an amount of lymphoid tissue as to make it resemble a single large Peyer's patch. Hawkins, on the other hand, is reservedly inclined to believe that the amount of lymphoid tissue does not bear any direct relation to the age of the individual, but states that only the examination of a large number of normal appendices would warrant any definite statements on this subject. Another histological peculiarity of the appendix not without significance is the relatively extensive epithelial surface that it presents. Under conditions of even slight irritation or erosion it thus affords a large field for the absorption of noxious products of bacteria.

The blood-supply of the appendix and the pathogenetic relationship that the bloodvessels and the arterial supply may bear to inflammation of that organ have been considerably studied. It is

unnecessary here to go into a detailed description of the blood-supply of the appendix, but I wish to make a few remarks upon the bloodvessels themselves. If I mistake not, it was Fowler and Van Cott who first directed particular attention to the important etiological rôle of the circulatory apparatus of the appendix in inflammation of that organ. These authors believe that the appendix is peculiarly exposed to vascular and nervous, and hence nutritional, disturbances, and base their opinion upon an examination of thirteen appendices sent by Fowler to Van Cott. The examination of these is said to have revealed in the vessels of the mesoappendix some form or other of obstruction to the blood current, either paravasculitis, perivasculitis, or endovasculitis; and these, it is believed, must have preceded the intense round-cell infiltration, the coagulation necrosis, and the purulent foci that they detected in the walls of the appendices. They also state that in several cases they detected a distinct chronic interstitial neuritis with extensive atrophy of the nerve-fibres resulting from hyperplasia of the endoneurium and perineurium. They argue from these that the real cause of the locus minoris resistentiæ admitting of bacterial invasion is not to be sought primarily in a trauma of the mucosa, but in a trophic disturbance of the appendix, the result of chronic vascular lesion, or chronic nerve lesion, or both, and that ulcerative processes in the appendix, while they may be increased by bacterial invasion, may nevertheless owe their origin to these trophic conditions. These opinions have been subjected to critical investigation by several observers, and have commonly not been confirmed. In particular Breuer, at the instigation of Nothnagel, carefully examined thirty appendices, some of which had been removed at necropsy and some by operation, among the latter there being instances of acute and chronic appendicitis. By means of carefully performed injection of the arteries he first of all determined that the vascular supply of the appendix is not a terminal one, such as obtains in the brain, spleen, kidney, etc., but that a not inconsiderable collateral blood-supply comes from the adjoining cæcal vascular area. Furthermore, it was determined that these anastomosing arterial branches course partly in the mucosa, partly in the muscularis, and partly directly beneath the

serosa. But, as the pertinent result of his investigations, Brener was unable to detect vascular alterations of the constancy and extent described by Fowler and Van Cott. In cases of chronic inflammation of the appendix the larger arteries of the mesoappendix were regularly intact, even when surrounded by hyperplastic connective tissue. Not only this, but the smaller vessels, the arteries and veins, of various areas of the wall of the appendix revealed but rarely pathological alterations. For instance, in areas in which the entire mucous membrane was entirely converted into cicatricial tissue, there were evident but slight thickening, endothelial proliferation of the intima, etc.—changes that it is asserted are detectable in every cicatricial tissue and in instances of normal involution of the appendix. In cases of acute suppurative appendicitis the alterations of the vessels were more common, but they were limited to the acutely inflamed region and its immediate vicinity. As the result of my own investigations in this particular, I believe there can be no question that Van Cott is correct in asserting the presence of these vascular alterations in some presumably normal and in some diseased appendices, but I also believe that he erroneously interprets their significance and overestimates their importance.

In a considerable number of presumably normal appendices removed at necropsy the thickness of the vessel walls of the appendix as well as of the mesoappendix impressed me forcibly. In many of these the deviations from the normal were almost exclusively confined to the muscular coat. In not a few instances, however, there was distinct proliferation of the intima. These alterations, particularly the endothelial proliferation, were more common in the diseased appendices examined. In some of the appendices that had been the seat of recurring attacks of inflammation the thickening of the vessel walls was very apparent. In some of the acute cases the endothelial proliferation was equally conspicuous. In other appendices—those which for a greater or less time had been the seat of chronic inflammation, and which had more recently suffered an acute exacerbation—alterations of all the walls were evident. Some interstitial connective tissue overgrowth was also occasionally detected in and about the nerves of the mesoappendix, but by no means as constantly and regularly as indicated by Van

Cott. As a result of my investigations, therefore, I ascribe considerable pathogenetic significance to the condition of the arteries, but much less to that of the nerves. However, excluding instances of thrombosis and embolism of the chief appendicular vessels or their branches, and obstruction of the blood-supply by means of torsions, angulations, or contracting bands of connective tissue compressing the vessels, I do not believe that ulceration of the wall of the appendix can with reason be ascribed to arterial alterations. I believe, though, that the precarious blood-supply of the appendix may with justice be held at least partly answerable for the disastrous consequences to the appendix of causes there provocative of inflammation, but which in other portions of the intestinal tract remain inoperative. I believe, also, that in case the blood-supply of the appendix becomes very defective, by torsion, angulation, etc., conditions obtain in the appendix that render the common exciting causes of ulceration (to be referred to later) very prone to produce their deleterious effects.

The indications of involution of the appendix demand careful consideration, and the proper interpretation of suggestive alterations is frequently a matter for judicious discrimination. Particular investigation of this question has been made, among others, by Ribbert and Zuckerkandl abroad, and by Piersol in this country. Ribbert examined four hundred appendices obtained at necropsy and determined that ninety-nine (25 per cent.) presented evidences of retrogressive atrophic alterations without indications of previous inflammation. These alterations were, therefore, interpreted as evidences of involution. Zuckerkandl investigated three hundred and twenty-three appendices and detected in fifty-five evidences of obliteration of the lumen—therefore in 23.7 per cent. Of one hundred appendices from persons over twenty years of age, examined by Ribbert, thirty-two presented these evidences of retrogression. The obliteration of the lumen was commonly but partial; it was complete in but 3.5 per cent. In one-half of the cases the distal quarter was closed; in one-half of the remaining cases the obliteration affected between one-quarter and three-quarters of the entire length of the appendix. In 14 per cent. of Zuckerkandl's cases the obliteration of the lumen extended throughout the length

of the organ; in the remaining 9 per cent. the obliteration was but partial. There can be no question that these evidences of retrogression are common. In the presumably normal appendices removed at necropsy that I examined they were found in almost one-fourth of the cases. They were usually confined to the tip and to a small portion of it. Exceptionally they implicated about one-fourth, or a little more, of the entire length of the organ. In these cases the remainder of the organ presented no recognizable histological deviations from the normal. These indications of involution were also evident in appendices manifestly the seat of inflammatory disease. In these latter cases also these involuntary alterations were limited to the distal end of the organ. I cannot recall an instance in which they implicated as much as one-half of the appendix. In all of these cases, however, the inflammatory character of the associated alterations was beyond question.

The exact nature of these involutionary alterations is still a matter of more or less conjecture. Ribbert states that in the appendices undergoing these changes three zones can usually be recognized: A central zone, more or less rich in cells; from this there is a gradual transition to the second zone, which is poor in cells and made up largely of connective tissue. Third, the muscular coat. The inner zone corresponds to the former mucous membrane; the middle to the submucosa. The former presents at times a small, narrow slit indicative of the former lumen. Careful examination of this, however, will often reveal a few strands of delicate connective tissue traversing it from side to side. These are readily torn, and thus likely to escape detection. Ribbert believes that the symmetry of the process, its progression from the distal to the proximal end, the absence of irregularities, of cicatricial tissue, and other indications of previous inflammatory conditions justify the assumption that the process is involutionary in nature. This is also indicated by the absence of degenerative alterations of the mucous membrane—the change being rather one of gradual atrophy. I believe, with Zuckerkandl, that the submucosa plays a most important rôle in these alterations, and that the changes in the other coats follow those of the submucosa. Piersol concurs in this opinion, stating that "changes within the submucosa inaugurate

the process leading to the retrogression of the appendix and precede the alterations affecting the mucosa." Defective nutritive supply is doubtless the basis of the process. As regards the rôle that these involutionary changes play in the causation of appendicitis, it suffices for the present to state that they are to be considered as indicative of a locus minoris resistentiæ, which permits of the more ready operation in the appendix, than in other portions of the intestinal tract, of the exciting causes of appendicitis.

The foregoing, then, are the factors that predispose the appendix to attacks of inflammation. They acquire their pathogenetic significance because they interfere with the proper and thorough drainage of the organs; because they reduce the capability of the organ to resist the influences of various morbific agencies; because of the facility with which nutritional disturbances may be engendered, and because of the relatively large surface presented for the absorption of the toxic products of bacteria that find their exit from the appendicular lumen retarded or prevented. Bearing in mind these facts when we institute a study of the pathogenesis of appendicitis, it is immediately patent that no one factor alone can be held accountable for the development of all cases of the affection. On the contrary, the above-mentioned anatomical and physiological peculiarities of the appendix render that organ less resistant to the well-known morbific agencies provocative of inflammation in other portions of the body. In individual instances one or the other of these peculiarities predominates over, and thus assumes a pathogenetic significance disproportionate to the others.

The exciting causes of appendicitis do not differ from those that induce inflammation in other regions of the body. As is well known, the most common causes of inflammation are mechanical and chemical irritation and bacteria. In a given case of inflammation it is often difficult to distinguish sharply between these etiological factors, particularly between the action of chemic irritants and bacteria. In this respect what is true elsewhere in the body is true also as regards the appendix. As a basis of a discussion of the pathogenetic rôle of bacteria in appendicitis, the results of my bacteriological investigation of certain appendices removed by operation may be detailed. The total number of appendices ex-

amined bacteriologically was two hundred and one, and all were removed during 1898. The first inoculation in each case was made from the lumen of the appendix after aseptic incision of its wall, and the inoculation was always made from the seat of most manifest disease. At times, as above narrated, inoculations were always made from the exudate on the peritoneal surface of the appendix, from the free pus in the peritoneal cavity, and from the drainage fluid subsequent to operation. The results of these latter inoculations in no case differed from those of the primary inoculations from the lumen of the appendix. Inoculations in the first place were made into bouillon or agar tubes, or both. Subsequently Petri-dish cultures were made, and later various other inoculations, for the purpose of fully establishing the identity of the bacteria under investigation. At times, also, cover-slip preparations were made directly from the pus or other contents, or from the exudate on the peritoneal surface of the appendix. For many reasons, however, these were not made as frequently as is desirable. Of the two hundred and one appendices examined bacteriologically, ninety-four were instances of acute appendicitis and one hundred and seven of chronic appendicitis. The results of the examinations may be tabulated as follows:

Acute appendicitis:		
Bacterium coli commune alone	69 cases,	73.4 per cent.
Bacterium coli commune and staphylococcus pyogenes aureus	13 "	13.85 "
Bacillus pyocyaneus alone	6 "	6.4 "
Staphylococcus pyogenes albus alone	3 "	3.2 "
Bacterium coli commune and streptococcus pyogenes	1 "	1.05 "
Staphylococcus pyogenes aureus alone	1 "	1.05 "
No growth	1 "	1.05 "
Total	94 cases,	100.00 per cent.

With reference to the various forms of acute appendicitis the results of the bacteriological examinations were as follows:

Acute catarrhal appendicitis:

Bacterium coli commune alone	3 cases.

Acute interstitial appendicitis:

Bacterium coli commune alone	11 "
Bacterium coli commune and staphylococcus pyogenes aureus	4 "
Bacillus pyocyaneus alone	2 "
Total	20 cases.

Acute ulcerative appendicitis without perforation:

Bacterium coli commune alone	27 cases.
Bacterium coli commune and staphylococcus pyogenes aureus	3 "
Bacillus pyocyaneus alone	3 "
Staphylococcus pyogenes aureus alone	1 case.
Total	34 cases.

Acute ulcerative appendicitis with perforation:

Bacterium coli commune alone	17 cases.
Bacterium coli commune and staphylococcus pyogenes aureus	4 "
Staphylococcus pyogenes albus alone	3 "
Total	24 cases.

Gangrenous appendicitis:

Bacterium coli commune alone	11 cases.
Bacterium coli commune and staphylococcus pyogenes aureus	2 "
Bacterium coli commune and streptococcus pyogenes	1 case.
Bacillus pyocyaneus	1 "
No growth	1 "
Total	16 cases.

The following were isolated from the cases of chronic appendicitis:

Bacterium coli commune alone	96 cases,	89.71 per cent.
Bacterium coli commune and staphylococcus pyogenes aureus	5 "	4.70 "
Bacillus pyocyaneus alone	1 case	0.93 "
Staphylococcus pyogenes aureus alone	1 "	0.93 "
Bacterium coli commune and bacillus prodigiosus	1 "	0.93 "
No growth	3 cases	2.80 "
Total	107 cases,	100.00 per cent.

The results of these bacteriological investigations present certain features of interest. It should be mentioned that in those instances in which cover-slip preparations of the contents of the appendix and of the periappendicular pus, etc., were examined the results of such examinations did not differ from the results obtained by culture methods. In this respect my investigations are somewhat at variance with those of other observers. It has been asserted by quite a few investigators in this field that appendicitis is the consequence of a mixed infection—that it rarely results from a mono-infection. The reason that bacteria other than the bacterium coli commune are cultivated in cultures with difficulty is said to lie in the fact that the latter named proliferates much more luxuriantly than do the others, and thus outgrow them. Tavel and Lanz first directed attention to the frequent implication of several forms of bacteria in the production of appendicitis, and these authors were soon followed by Barbacci, and since then by many others, among them Welch and others in our own country. In but twenty of my two hundred and one cases was such mixed infection demonstrated by the methods of investigation pursued. These comprised fourteen acute cases (15 per cent.) and six chronic cases (5.6 per cent.). While I am free to admit that the systematic examination of cover-slip preparations of the contents of all diseased appendices, as well as of the exudate on the peritoneal surface of such diseased appendices, and of free pus in the abdominal cavity, might have resulted in the detection of the associated presence of two or more varieties of bacteria in a larger number of instances, I am, nevertheless, confident that the bacterium coli commune plays by far the predominant rôle in the bacterial origin of appendicitis. While this is but negative evidence as regards the possible presence of other bacteria, it is, nevertheless, suggestive that the bacterium coli commune was found in one hundred and eighty-five (92 per cent.) of the cases. When to this we add the fact that the conditions in the appendix are most favorable for the sudden and rapid increase in the virulence of a bacterium, innocuous or almost so in other portions of the intestinal tract, it seems to me that the predominating importance of this organism cannot be gainsaid. It cannot be denied that the bacterium coli commune, under

suitable conditions, proliferates most luxuriantly in culture media. Nor, on the other hand, can it be denied that the appendix is a most appropriate test-tube and the contents of the appendix most suitable media in which to cultivate a virulent growth of the bacterium coli commune. I believe, therefore, that as this organism outgrows other bacteria in artificial media, so also does it, under suitable conditions, assume a predominance in the appendix. This organ, as the intestine under normal conditions, contains a variety of bacteria, but under conditions favorable for the development of appendicitis, the bacteria coli commune rapidly becomes the predominating and most virulent organism. These statements do not eliminate from consideration other bacteria as etiological factors in inflammatory affections of the appendix. A number of other organisms have been found sufficiently often by various investigators as well as by myself, and their virulence has been repeatedly tested to indicate their importance in the production of appendicitis. Undoubtedly in certain cases these other bacteria do contribute to cause the disease; in other instances they are doubtless the sole etiological factors. Admitting this, I wish in this connection merely to record my belief in the predominating importance of the bacterium coli commune.

In discussing the pathogenesis of appendicitis, particular attention must be devoted to the significance of foreign bodies and fecal concretions. It is well remembered that early in the development of our knowledge concerning appendicitis the origin of the disease was commonly attributed to the presence in the appendix of various foreign bodies, such as cherry-stones, grape-seeds, seeds of various other fruits, pins, needles, hair, bits of bone, gallstones and the like. Of recent years, as a result of more careful investigation, it has become evident that many of the bodies considered seeds of various fruits were in reality but fecal concretions, the misconception as to their real nature being due to the resemblance that they bore in size and shape to the various objects for which they were mistaken. Undoubtedly foreign bodies other than fecal concretions do gain access to the appendix, and in the event of the organ subsequently being the seat of inflammation it is but natural to attribute an etiological rôle to such foreign body.

Of the foreign bodies found in the appendix a pin is, curiously, one of the most frequent.

While in the light of recent experimental and other scientific research the views formerly maintained with regard to the rôle of various foreign bodies in the production of appendicitis has largely been abandoned, the relation of what we now know to be fecal concretions to the development of appendicitis still demands careful consideration. That fecal concretions may be borne in the intestinal tract and in the appendix without inducing any important pathological process is well established as the result of the investigations of a considerable number of observers, and is being constantly confirmed by all who have occasion to see much necropsy work. On the other hand, that fecal concretions are associated with a considerable number of cases of appendicitis is equally well established. They are found in the appendix more often when it is inflamed than when it is normal, and more commonly in cases of acute ulcerative appendicitis with perforation and in gangrenous appendicitis than in any of the other acute varieties. They are also rather frequent in cases of chronic appendicitis. They are noted in seventy-four (16 per cent.) of my cases. There can, however, be no question that they are more commonly associated with cases of acute appendicitis than these figures would indicate. It not infrequently happens in cases of ulcerative appendicitis with perforation and in gangrenous appendicitis that at the time of the operation the fecal concretion has already been discharged from the lumen of the appendix, and is not detected during the operative manipulations, or, being detected, is not preserved. I thus believe that in ulcerative and gangrenous appendicitis fecal concretions play an important rôle.

It is not pertinent to discuss at present the question of the formation and chemical composition of these fecal concretions. It suffices to state that in contradistinction to the older view, according to which these concretions were formed within the intestine, and, being formed, found their way into the appendix, it is now generally considered that they are formed within the appendix itself. Fecal matter becomes inspissated, and forms a nucleus about which

are deposited in successive layers inspissated mucus, desquamated epithelium cells, at times leucocytes, etc.

Considering, now, the relative rôles played by bacteria and fecal concretions in the causation of appendicitis, it seems evident to me that appendicitis is almost without exception an infectious process; that the inflammations of the appendix are the result of the activities of bacteria. The rôle played by fecal concretions is considered subsidiary to that of bacteria. Such being the case, the queries naturally suggest themselves, Why is it that bacteria normally present in the intestinal tract are provocative of such serious pathological alterations in the appendix? And why is it that fecal concretions innocuous in the intestinal tract are associated with inflammatory affections of the appendix? The reasons for these are to be found in the anatomical and physiological peculiarities of the appendix, of which mention has been made above. These, on the one hand, decrease the capability of the appendix to resist the influences of various morbific agencies, and on the other afford opportunities for the rapid increase in virulence of bacteria contained within the organ. Disease is due not alone to the virulence of the determining cause, be it bacteria, trauma, or other cause, but is dependent to a considerable degree upon the predisposition of the individual, upon the vitality, and the power of resistance of the part attacked. Thus it is with the appendix. Because of the anatomical and physiological peculiarities above detailed, morbific agencies readily overcome by the normal physiological activities of the intestinal tract are capable, when present in the appendix, of inducing the most deleterious consequences.

To recapitulate, then: The factors that operate to render the appendix less resistant than other portions of the intestinal tract to the onslaughts of bacteria and other determining causes of inflammation are several. Of prime importance is the precarious blood-supply of many appendices. The blood-supply is defective not alone because of the manifest alterations frequently demonstrable in the walls of the bloodvessels, but also because of the liability of the occurrence of partial or complete obstruction of the blood-channels as a result of angulations, torsions, etc. Of importance in a limited number of cases is doubtless also alterations in the

nerves supplying the appendix; but, as already stated, it is believed that they are not of such great importance as has been suggested by Fowler and Van Cott. Finally, in this connection the evidences of retrogression of the organ are not without significance. The factors that in the appendix give rise to increase in the virulence of bacteria normally present in the intestine are such as interfere with thorough drainage of the organ. Defective drainage may supervene when for any reason the appendix is so situated that it cannot be readily emptied; when its lumen is constricted either externally by bands of cicatricial connective tissue, tumor formations, etc., or by cicatrices of its walls, or by obstructions within its lumen—as, for instance by fecal concretions of foreign bodies; or when the muscular coat of the organ is no longer capable of active peristalsis, as is likely to be the case when the appendix is itself diseased or bound down by adhesions. Congestive disturbances of the appendix or of the cæcum may cause such swelling of the mucous membrane as to lead to approximation of the apposed surfaces in the appendix, or occlusion of the outlet of the organ, and thus effectually prevent drainage. Of considerable significance with reference to this question of drainage in the etiology of appendicitis are certain of the anatomical and physiological peculiarities of the organ above alluded to. Of these I may mention the size and shape of the mesoappendix, the excessive length as compared with the calibre of the lumen of the appendix, and Gerlach's valve. As further conducive to imperfect drainage are the already mentioned torsions and angulations, which interfere not alone with the blood-supply but also with thorough drainage. Fecal concretions are capable of at least a twofold action. They may not only occlude the lumen of the appendix, and effectually prevent drainage, but they may also, by attrition, the result of constant or intermittent peristalsis, cause erosions of the mucous membrane, and thus reduce the power of the organ to reduce the attacks of bacteria or their toxins. When of small size these concretions are doubtless often innocuous, but when they have attained a considerable size and are no longer capable of being extruded they may engender the most disastrous results.

The preponderating importance of defective drainage in the

pathogenesis of appendicitis is sufficiently evident from an examination of a large number of diseased appendices, but it finds additional confirmation from the clinical and pathological observation of analogous intestinal conditions and from some experimental investigations. Increase in the virulence of bacteria has been found not only in the appendix when its lumen has been obstructed, but also in cases of intestinal obstruction and strangulation and in various congestive and diarrhœic conditions, and even in cases of marked and long-continued constipation. The experimental investigations of Ronx, Roger and Josué, Dielafoy, and of Frazier are also corroborative of this. The last named states as a result of his experiments on rabbits that "of all the etiological factors that enter into the pathogenesis of appendicitis imperfect drainage of the organ plays the most conspicuous part," and "as a result of the interference with drainage the innocuous bacillus coli communis is converted into a virulent organism." Lastly of importance is the relatively large extent of mucous membrane presented by the appendix and the large amount of lymphoid tissue not only in the neighborhood of Gerlach's valve but also scattered throughout the walls of the organ. The mucous membrane, particularly if it be eroded, thus presents a very large surface for the ready invasion of bacteria and the absorption of their noxious products. This latter is of especial importance in the production of gangrene of the appendix, which is often the result of infective thrombosis or embolism following erosion or ulceration of the walls of the appendix.

From the foregoing, therefore, it is evident that no one factor can be held answerable for the production of all cases of appendicitis. Although the disease is almost without exception the consequence of micro-organismal infection, it is rather of complex pathogenesis, and no one morbific agent is provocative of all attacks. It is because of the anatomical and physiological peculiarities of the appendix that factors innocuous in the intestine, or morbific agents capable of being successfully counteracted by the physiological activities of the intestine, become in the appendix of heightened virulence and engender the most disastrous consequences.

REPORT OF 460 CASES OF APPENDICITIS OPERATED UPON AT THE GERMAN HOSPITAL IN TWO YEARS ENDING JANUARY 1, 1899.

By JOHN B. DEAVER, M.D.

Compiled by G. G. Ross, M.D., and S. W. Dougherty, M.D.,
members of the hospital staff.

[Read April 5, 1899.]

Operations.

Acute,	264	Died,	46	Mortality,	17.4 per cent.
Chronic,	196	"	0	"	0
Total cases,	460	Total deaths,	46	Mortality,	10 per cent.

Males, 307 Females, 153

Ages.

Birth to 5 years	1	30 to 35 years	72
5 to 10 "	14	35 to 40 "	35
10 to 15 "	29	40 to 45 "	20
15 to 20 "	56	45 to 50 "	4
20 to 25 "	125	50 to 60 "	16
25 to 30 "	86	60 to 70 "	2

Youngest, 4 years. Oldest, 68 years.

Number of Attacks.

148 had	1 attack	2 had	8 attacks
89 "	2 attacks	2 "	9 "
52 "	3 "	4 "	10 "
20 "	4 "	1 "	12 "
18 "	5 "	2 "	16 "
16 "	6 "	2 "	20 "
6 "	7 "	73 "	Many attacks

1 had constant pain for 15 years
2 " " " " 3 "

1 had constant pain for 3 months
312 had more than one attack.

Condition of Bowels.

Constipation	249
Diarrhœa	42
Normal	39
Alternate constipation and diarrhœa	11

Tenderness on pressure in	433
Rigidity of the overlying abdominal wall	426
Dulness on percussion	80
Nausea and vomiting	300
General distention	91
Mass	29
Œdema of the overlying abdominal wall	12

Time of Operation.

After attack	281	Of these	3 died
During attack	165	"	43 "
Not stated	14		

Localized peritonitis	68	Of these	6 died
General peritonitis with free pus	20	"	17 "
Localized abscess	140	"	26 "
Adhesions	201	"	6 "
Gangrene	78	"	39 "
Perforation	88	"	24 "
Cæcum or bowel involvement	84		
Pus in appendix	66		

Of 18 cases operated within 24 hours,				1 died	33 per cent.
" 6 "	"	"	36 "	1 "	20.0 "
" 24 "	"	"	48 "	9 "	26.4 "
" 35 "	"	"	3 days,	5 "	
" 27 "	"	"	4 "	5 "	
" 13 "	"	"	5 "	2 "	
" 11 "	"	"	6 "	2 "	
" 29 "	"	"	7 "	4 "	
" 7 "	"	"	8 "	1 "	
" 14 "	"	"	10 "	3 "	
" 5 "	"	"	11 "	0 "	
" 2 "	"	"	12 "	0 "	
" 2 "	"	"	13 "	0 "	
" 14 "	"	"	14 "	0 "	
" 13 "	"	"	21 "	3 "	
" 1 case	"	"	24 "	0 "	
" 1 "	"	"	25 "	1 "	
" 2 cases,	"	"	35 "	0 "	

Of	1 case	operated	within	4 weeks,	0 died
"	2 cases,	"	"	5 "	1 "
"	1 case,	"	"	6 "	0 "
"	1 "	"	"	8 "	0 "

Not stated in 15 cases.

Of	24	acute cases	operated on	within	36 hours,	2 died	8.3 per cent.
"	59	"	"	"	3 days,	14 "	23.7 "
"	27	"	"	"	4 "	5 "	18.5 "
"	13	"	"	"	5 "	2 "	15.3 "

FATAL CASES (ACUTE) APPENDIX OPERATIONS.

Name.	Time between onset of present attack and operation.	Pathological conditions.
Aldrich,	7 days; two operations; only attack.	Appendix gangrenous; large abscess with pus free in peritoneal cavity.
Beers.	21 days; one previous attack.	Appendix gangrenous and sloughed off; general peritonitis at time of operation.
Brooks.	4 days; only attack.	Appendix perforated and gangrenous; large abscess; localized plastic peritonitis.
Coe.	18 hours; only attack.	Appendix perforated; localized collection of pus; abscesses of both lungs; post-mortem showed also acute endocarditis.
Dennison.	5 days; only attack.	Appendix perforated and gangrenous; large quantity of greenish pus; gangrenous tissues surrounding appendix.
Gaskill.	One attack 2 years before; 24 hours.	Gangrenous appendix.
Hopkins.	Three mild attacks before; 3 days.	General peritonitis; appendix gangrenous; large abscess.
Howell.	1½ days.	Patient had valvular heart disease; gangrenous appendix.
Kelly.	21 days.	General peritonitis; large abscess; appendix perforated; gangrenous.
Maguire.	One previous attack; 3 days.	Appendix gangrenous and perforated; abscess; general peritonitis.
Martin.	One previous attack; 10 days; two operations.	Large abscess; later another operation, and two more pus collections and gangrenous appendix found; peritonitis.
McCartney.	10 days; only attack.	Gangrenous appendix; pus free in peritoneal cavity; purulent peritonitis.
McLaughlin.	Several mild attacks; 7 days.	Gangrenous appendix distended with pus; cæcum and small bowel involved.
Mousel.	2 days.	Gangrenous appendix; infracolic and retrocolic abscess; plastic peritonitis; acute nephritis.

Name.	Time between onset of present attack and operation.	Pathological conditions.
Ottinger.	Mild attacks two years before; 14 days.	Eleven large abscesses; cæcum perforated and feces discharged into peritoneal cavity; purulent peritonitis.
Roepke.	4 days.	Appendix gangrenous and perforated; large abscess; bowel shows gangrenous areas; general purulent peritonitis.
Robbins.	5 days.	Appendix gangrenous and perforated; large abscess.
Roser.	3 days.	Acutely inflamed appendix; general peritonitis.
Smith	Mild pain for one year; 4 days; two operations.	Large postcæcal abscess; fecal vomiting developed; second operation showed bowel matted together by dense adhesions, causing partial obstruction; patient died from shock.
Shinn.	14 days.	Large abscess; general peritonitis.
Sweeney.	21 days.	Appendix gangrenous and perforated; large abscess; purulent peritonitis.
Amos.	24 hours.	Appendix perforated and gangrenous; abscess; purulent peritonitis.
Brooks.	Four mild attacks; 14 days.	Appendix acutely inflamed and perforated; retrocæcal abscess; purulent peritonitis.
Gobright.	Several previous attacks; 2 days.	Perforation; large quantity of pus; purulent peritonitis.
Kemberly.	One previous attack; 2 days.	Gangrenous appendix and omentum; died from pylephlebitis day after operation.
Kircher.	4 days.	Acutely inflamed appendix; developed lobar pneumonia, and died.
Lunning.	10 days; two operations.	Large abscess drained; appendix removed and a secondary abscess drained. Autopsy showed abscess of liver.
Luffeberry.	3 days.	Gangrenous appendix which had separated; large quantity of pus free; purulent peritonitis at time of operation.
Martin.	Two previous attacks; 2½ days; two operations.	Abdominal walls infiltrated; large abscess with involvement of cæcum; appendix perforated and gangrenous; cæcum gangrenous; purulent peritonitis.
McDonald.	5 weeks.	Large appendiceal abscess and pyosalpinx.
Mitchell.	6 days.	Appendix gangrenous and perforated; large abscess; purulent peritonitis.
Murphy.	7 days.	Appendix sloughed off and gangrenous; large abscess; purulent peritonitis.
Pettit.	3 days.	Perforated and gangrenous appendix; postcæcal abscesses; purulent peritonitis.

Name.	Time between onset of present attack and operation.	Pathological conditions.
Rayes.	2 days.	Acutely inflamed appendix; abscess in broad ligament; infection from purulent peritonitis.
Rhodes.	Many attacks; 7 days.	Appendix gangrenous and perforated; large abscess; purulent peritonitis.
Schroeder.	2 days; first attack.	Gangrenous and perforated appendix; large abscess; pus in pelvis; purulent peritonitis.
Shallhorn.	25 days.	Septic on admission; gangrenous and perforated appendix; large abscess. Autopsy showed pylephlebitis.
Smith.	4 days.	Appendix perforated and gangrenous; large abscess; purulent peritonitis.
Windt.	6 days.	Appendix and mesoappendix gangrenous; purulent peritonitis.
Weth.	One previous attack; 2 days.	Gangrenous appendix; abscess; purulent peritonitis.
Prescott.	2 days.	Abscess; cæcum involved.
Merkinger.	14 days.	Abscess; purulent peritonitis.
Ottey.	4 days.	Universal intestinal adhesions; purulent peritonitis.
Wilson.	....	Perforation; gangrene; abscess.
Mugdoss.	.....	Perforation; gangrene; abscess.
Keister.	.....	Gangrene; perforation; abscess.

Secondary Operations.

1897.			
Johnson.	Acute.	Recovery.	Free pus.
John Snyder.	Acute.	Recovery.	Lumen filled with débris.
May Carey.	Acute.	Recovery.	Lumen collapsed.
1898.			
A. Schoerwolf.	Acute.	Recovery.	
Schmecker.	Chronic.	Recovery.	
Aldrich.	Acute.	Death.	Large abscess with free pus.
Martin.	Acute.	Death.	Large abscess; second operation showed two more pus collections.
Smith.	Acute.	Death.	Second operation showed occlusion of bowel.
Luenning.	Acute.	Death.	Second operation, drainage of second abscess; abscess of liver.
Alf. Smith. (Drexel Home.)	Acute.	Recovery.	Abscess; second operation to remove organ.

Kliner.	Acute.	Recovery.	First attack; traumatism; second operation to remove organ.
J. Marti.	Acute.	Recovery.	Second operation.
J. Freetog.	Acute.	Recovery.	Second operation.

Primary, 251	Deaths, 42	16.7 per cent.
Secondary, 13	Deaths, 4	30.7 per cent.

Histories of Patients who Died from Acute Appendicitis.

Acute Appendicitis.

G. M. A., aged thirty-two years. Died. Admitted on the seventh day of attack. General abdominal pain and tenderness most marked in right iliac fossa. Vomited. Had a fluctuating mass in right side. Pus free in abdominal cavity. Appendix gangrenous at bottom of cavity; had perforated. Did well for one week, when temperature went up, and secondary operation was performed, removing organ. Developed nephritis, and died with temperature of 107°.

Post-mortem. Infracolic and retrocolic abscess, localized plastic peritonitis, fatty infiltration of liver and pyelitis. Both kidneys show evidence of nephritis.

Mrs. B. Two attacks. Admitted with general peritonitis; last attack began three weeks ago; between the two attacks had constant trouble with bowels, alternating diarrhœa and constipation. Had attack of jaundice at onset of attack. General abdominal pain, tenderness and distention.

Operation. General abdominal adhesions. Appendix sloughed off, gangrenous. She died of shock a few hours after leaving the table.

A. B., aged fifty-five years. Admitted to the German Hospital. Four days sick when admitted. Had a mass in right iliac fossa, tender, and about the size of a large orange.

Operation. Large abscess. Organ gangrenous and perforated.

Post-mortem Diagnosis. Croupous pneumonia right lower lobe. Chronic endocarditis. Slight plastic peritonitis. Acute nephritis (left.) Right kidney contracted, with abscess cavities which were probably tubercular.

F. C., aged nineteen years. Attack began suddenly on the day before admission. On admission about eighteen hours (12.30 A.M.) after onset of attack, temperature was 101.2°, pulse 116; severe pain and tenderness right iliac fossa, vomiting and rigidity. Operated 3.30 P.M. same day. Free pus escaped; large abscess; appendix perforated, lying postcæcal. Patient developed rash, and was transferred to isolating house.

Diagnosis. Measles. Developed croupous pneumonia and septic abscess of lungs.

Post-mortem Diagnosis. Infracolic and retrocolic abscess, localized plastic peritonitis. Double abscess lungs. Double lobar pneumonia of both infe-

rior lobes, acute endocarditis, acute splenic tumor, acute nephritis and right-sided pyelitis. Typhoidal ulceration of ileum and colon.

H. D., aged forty-five years. Admitted to the German Hospital. Ill five days with attack before admission. General pain and tenderness and vomiting. Large abscess. Patient said he felt all right and wanted to go home.

Operation. Omentum red and adherent. Large quantity of greenish pus. Appendix postcæcal, perforated gangrenous, as was surrounding tissues. Patient had chill on fourth day after operation, and died. Temperature 97.4°.

C. G. Attack two years ago, and as it was slight in character operation was deferred. Present attack began at 4 A.M. on November 9, 1898, cramp, constipation, vomiting, general tenderness soon becoming localized in right iliac fossa; rigidity. Temperature on admission 98.4°; pulse 88.

Operation November 9, 1898. Appendix postcæcal, gangrenous and tightly adherent. No pus.

13th. Wound inflamed; tension on stitches relieved by incision. Calomel and asafœtida enema gave relief and patient spent a quiet night. Early next morning patient suddenly had a convulsive shudder, clasped hand over his heart, and died. Appendix contained bacillus pyocyaneus.

R. H., aged twenty-five years. Three previous attacks of so-called intestinal colic. Admitted August 29, 1898; taken on the Thursday before that date. Usual symptoms, excepting that the pain was most severe on the left side. Most marked one inch above Poupart's ligament. By Sunday the pain was localized on right side, accompanied by tenderness, rigidity, etc., although left rectus was still more rigid and very tender. Vomitus had fecal odor. Abdomen very much distended.

Operation. Abdominal walls infiltrated; large abscess; dense adhesions. Appendix gangrenous. General peritonitis.

E. H., aged twenty-one years. Valvular heart disease. Admitted October 21, 1898. Attack began Thursday prior to admission; grew rapidly worse. Usual symptoms and location (admitted day after onset). General distention and tenderness and rigidity. Vomiting. No constipation.

Operation same afternoon. Appendix gangrenous. No pus. Closed with gauze drainage. Temperature 102° on admission; pulse 116.

T. K. Sick three weeks prior to admission. General abdominal pains, vomiting and constipation one week ago; trouble centred in right side. Large mass in right side on admission, tenderness and dulness and marked distention of abdomen.

Operation. Large abscess. Many adhesions. Appendix gangrenous and perforated. General peritonitis.

T. M. Two attacks; last one began on Wednesday before date of admission, October 9, 1898. Slight at first, but gradually becoming worse, accompanied by tenderness, rigidity, pain and vomiting, constipation. General abdominal distention.

Operation night of admission. Had abscess and gangrenous appendix; also perforation. Patient died same night, probably of shock.

T. T. M., aged thirty-three years. First attack six weeks ago. Present attack began ten days before admission, accompanied by usual symptoms. Painful mass in right iliac fossa, marked tenderness and rigidity and dulness. Fluctuation.

Operation on day of admission. Large abscess; appendix not searched for, as abscess-wall was so firm. Second operation: Secondary abscess, in which the appendix was found. Search for appendix discovered two more collections of pus, one in the pelvis. Intravenous transfusion, salt solution. Abdomen became distended, and patient died of peritonitis.

S. McC. One attack, began seven days prior to admission. Usual onset and symptoms. Morphine had been administered and patient grew steadily worse. General distention; rigid, tender, painful abdomen. Pulse 118; temperature 101.2°. Operated on third day after admission and when symptoms had improved. Free pus in abdomen. Appendix gangrenous and in three sloughs. Patient died of purulent peritonitis.

P. McL., aged thirty-four years. Numerous slight attacks. Present attack began seven days ago. Usual symptoms, but not marked.

Operation. Appendix posteæeal and adherent. Appendix full of pus. Died same day.

P. M., aged fifty-seven years. Present attack two days before admission. Usual symptoms and great severity. Temperature 101.2°; pulse 112. Very sensitive right iliae fossa.

Operation same day. Appendix gangrenous. Developed peritonitis in six hours and died.

Post-mortem diagnosis: Infraeolic and retroeolie abscess. Sclerosis of aortie semilunaris. Pulmonary congestion. Fatty infiltration of liver. Acute nephritis. Plastic peritonitis.

J. O., aged twenty-five years. Attack two years ago. Two since then. Present attack began two weeks before admission. Marked, usual symptoms.

Operation. Large abscess Perforation into cæcum, with free discharge of feces into peritoneal cavity. General purulent peritonitis.

R. Admitted August 29, 1898. Present attack dates back to preceding Friday. Usual symptoms on right side, but extending to left side. Has a small tender mass in right iliae fossa.

Operation. Large abscess. General purulent peritonitis. Appendix gangrenous and perforated; dense adhesions. Bowel was involved in gangrenous proeess.

W. R., aged seventeen years. Admitted August 24, 1898.

19*th.* Attack began after violent exercise and injudicious eating. Usual symptoms; very severe. Constipation.

24*th.* On admission general abdominal pain and tenderness, most marked

on right side. Owing to flexion of leg his doctor had diagnosticated luxation of femur.

Operation. Appendix postcæcal, perforated, and a large abscess was present. Early gangrene of appendix.

E. R., aged fourteen years. Admitted to the German Hospital November 25, 1898. The first attack began three days before admission. Usual symptoms not severe.

Operation. Adherent appendix, thickened and acutely inflamed. Wound closed without drainage.

Post-mortem. General purulent peritonitis from streptococci infection; operation.

W. S. History of trouble in right iliac fossa for one year. Present attack of four days' duration. Usual symptoms. Dulness on right side, but no mass; tenderness, rigidity and distention.

Operation. Abscess behind cæcum. Abdomen distended; fecal vomiting. Second operation, bowel matted together by strong adhesions, causing partial obstruction. Patient died of shock.

D. S., aged twenty years. Sick in United States Camp for two weeks prior to admission. General pain; constipation and vomiting.

Operation Liberated large quantity of pus and cavity drained. General peritonitis.

J. S., aged twenty-eight years. First attack began three weeks ago. Usual symptoms, but severe; vomiting, which had been severe, ceased one week prior to admission, although tenderness and pain persisted. Large, painful mass in right iliac fossa.

Operation. Large abscess evacuated. Appendix gangrenous and perforated. Adhesions. Died of general purulent peritonitis.

C. A., aged twenty-seven years. First attack. Usual symptoms began day before operation.

Operation. Appendix perforated and gangrenous. General distention. Abscess. Died of general purulent peritonitis.

O. B. Troubled with cramps for two years. Four attacks, last one two weeks prior to admission. Usual symptoms. Had mass in right iliac fossa.

Operation. Appendix adherent; peritoneum highly inflamed and abdomen distended. Abscess.

Post-mortem. Retrocolic abscess. General purulent plastic peritonitis.

L. G., aged seventeen years. Several previous attacks. Present attack began two days prior to admission. Usual symptoms. General distention and tenderness. Abscess and purulent peritonitis (general).

J. K., aged thirty-three years. Two attacks, last one beginning two days prior to admission. Usual symptoms, of severe type. Very tender and rigid. Gangrenous appendix and omentum. Temperature shot up immediately after operation to 102°, and he had a chill while coming out of ether. No distention and very little pain. He developed marked jaundice. High

temperature of 102° persisted, with chills. Urine showed albumin, hyaline casts, blood-cells, and epithelium. Jaundice and gall-bladder tenderness persisted until patient died.

C. K., aged thirty-two years. One attack began four days prior to admission. Usual symptoms.

Operation. Appendix acutely inflamed. No abscess, gangrene, or perforation. Second day after operation patient had a chill and rise of temperature to 103.6°. He developed marked lobar pneumonia, from which he died.

L. W. First attack began ten days ago, with two exacerbations. Usual symptoms; large abscess when admitted.

Operation. Opened abscess. Second operation: Appendix removed and a large secondary abscess opened. Autopsy discovered a large abscess of liver. Pelvic adhesions, but no more pus.

B. L. First attack three days before admission. Usual symptoms, associated with headache. Symptoms steadily grew more severe, and he developed general peritonitis. Abdomen distended; general tenderness.

Operation same day. Free pus; gangrenous appendix, which had separated; fecal concretion loose in abscess-sac. Died of general purulent peritonitis.

G. M., aged thirty-seven years. Two previous attacks. Last one, very severe, began two or three days prior to admission. Distention and pain. Tenderness most marked over right iliac fossa. Abdominal wall œdematous.

Operation. Abdominal walls infiltrated. Large abscess evacuated. Appendix gangrenous and perforated. Cæcum involved. Died of general purulent peritonitis.

Mrs. McD., aged thirty years. One attack, extending over five weeks, with repeated exacerbations. Usual symptoms, becoming steadily more severe. Excessive tenderness.

Operation. Abscess; adhesions; appendix wrapped in mass of organized lymph. Pyosalpinx complicated case. Temperature remained high, 103°, with high pulse, 160. Peritonitis.

A. M., aged twenty years. Admitted to the German Hospital July 7, 1897. First attack began six days prior to admission. Usual symptoms, not becoming severe until day before admission. Tenderness exquisite in right iliac fossa; marked rigidity and slight distention, with dulness. Persistent vomiting.

Operation. Opened large abscess. Adhesions. Appendix gangrenous and perforated, fecal concretion in cavity. Died next day of peritonitis.

L. M., aged twenty-six years. Admitted July 26, 1897. Sick one week with first attack. Usual symptoms marked. Abdominal walls rigid, markedly tender, and a mass felt in right iliac fossa, which is dull on percussion.

Operation. Appendix adherent; abscess behind cæcum. Appendix

sloughed entirely off and gangrenous into mesoappendix. Died of general purulent peritonitis.

H. P., aged twenty-nine years. First attack three days' duration. Usual symptoms, except diarrhœa. General distention; tenderness and mass in right side; extreme rigidity.

Operation. Abscess. Appendix postcæcal, perforated and gangrenous. Died of general purulent peritonitis.

F. R., aged twenty years. Admitted to the German Hospital November 14, 1897. Usual symptoms, with some distention.

Operation. Appendix postcæcal and postcolic, very long, very highly congested. In removal the organ was cut and contents escaped into peritoneal cavity. Left ovary, tube removed for cyst, and pus in broad ligament. Patient died of septic peritonitis due to infection from abscess in broad ligament.

R., aged thirty-seven years. Admitted to the German Hospital June 8, 1897. Repeated attacks for fifteen years. Sick for at least one week. Usual symptoms, of severe type. Extreme tenderness and rigidity; slight distention.

Operation. Large abscess, not localized. Appendix adherent, gangrenous, and perforated. Second operation to relieve abdominal distention due to peritonitis. Died of shock and general purulent peritonitis.

S. O., aged twenty-four years. Admitted to the German Hospital July 13, 1897. Attack began two days before admission; usual symptoms; first attack. Marked tenderness and rigidity in right iliac fossa. No marked distention. Dulness right iliac fossa.

Operation. Large abscess. Appendix gangrenous and perforated; pus in pelvis. Died of general septic peritonitis, July 17, 1897.

J. S., aged forty-three years. Admitted to the German Hospital December 19, 1897. Attack began twenty-five days before admission. Usual symptoms. Generally septic when admitted.

Operation. Large abscess, postcæcal. Appendix gangrenous and perforated. Developed pyophlebitis, from which he died.

Post-mortem. Acute parenchymatous nephritis. Fatty infiltration and degeneration of heart. Hepatic pyophlebitis. Subhepatic and retrocæcal abscess. Slight atheroma of aortic arch.

T. S., aged fifteen years. Admitted to the German Hospital August 12, 1897. Attack began two days prior to admission. Usual symptoms and signs.

Operation. Two days after admission. Large abscess. Appendix gangrenous and perforated. Belly distended and pulse became weak. Died of septic peritonitis.

C. W., aged thirty-three years. Admitted to the German Hospital August 30, 1897. Six days prior to admission usual symptoms and signs. Rapid pulse and general peritonitis.

Operation. Appendix and mesoappendix gangrenous, perforation. Died of general purulent peritonitis.

L. W., aged twenty-eight years. Admitted to the German Hospital December 14, 1897 One previous attack. Present one began two days prior to admission; usual symptoms and signs, of severe type. Abdomen distended, and a mass can be made out in right iliac fossa.

Operation one hour after admission. Abscess appendix gangrenous. Death from general purulent peritonitis, December 18, 1897.

P. (Children's Hospital Case.) Abscess retrocæcal. Died of general sepsis.

W. O., aged thirty-seven years. Admitted to the German Hospital September 13, 1898. Patient walks into house under the influence of morphine; had painful belly, dull iliac fossa. Operation advised and accepted, since his doctor brought him for such.

Operation. Under anæsthesia a small cut is made through the right semilunar line, exposing an adherent omentum. Incision prolonged to five inches. Cæcum found bound down tightly, small loop of bowel adhering to one another throughout, five very dense adhesions, necessitating cutting, about twelve other firm ones were cut; to relieve all these it was necessary to expose the intestinal coil. Appendix, lying behind cæcum, had to be dissected out, then enucleated. Glass drainage introduced to pelvis, with one piece of iodoform gauze behind cæcum; had to empty small bowel before we could restore it; 30 c.c. saturated mag. sulph. sol. injected. Condition simply remarkably favorable for five days, when he suddenly grew delirious, septic, and finally died on eighth day of disease, of purulent peritonitis.

J. K. Large abscess. Patient died of general septic peritonitis.

W. Appendix perforated and gangrenous, and a large abscess had formed in right iliac fossa. Patient died of sepsis.

M. Appendix gangrenous and perforated. Large abscess. Patient died of sepsis.

K. Appendix gangrenous and perforated. A large abscess was present. Patient died of sepsis.

Histories of Patients who Recovered.

Case 1. K. Acute. One attack. Abscess; adhesions. Appendix gangrenous.

Case 2. J. Acute. Two attacks. Abscess; peritonitis; free pus in peritoneal cavity. Adhesions involving cæcum and small bowel. Appendix gangrenous and perforated.

Case 3. S. Acute. One attack. Abscess; peritonitis; free pus in peritoneal cavity; extensive adhesions. Appendix gangrenous and perforated. Structure of appendix entirely destroyed.

Case 4. S. Acute. One attack. Localized abscess. Appendix gangrenous and perforated. Lumen filled with inflammatory exudate.

Case 5. L. Acute. One attack. Mucous coat of appendix abraded; lumen contained fecal matter.

Case 6. A. N. Acute. One attack. Mucosa inflamed.

Case 7. S. Acute. Three attacks. Adhesions and inflamed mucous membrane.

Case 8. V. Acute. Two attacks. Mucous coat of appendix eroded; the muscular coat contained inflammatory deposit.

Case 9. A. Acute. Two attacks. Localized abscess. Lumen occluded with distention beyond seat of obstruction. Fecal concretion.

Case 10. A. C. Acute. First attack. Œdema of abdominal walls. Localized abscess and peritonitis. Appendix perforated at one place and occluded at another.

Case 11. P. Y. Acute. Five attacks. Had adhesions; appendix gangrenous and occluded at one place.

Case 12. P. Acute. Two attacks. Adhesions and abscess. Lumen contained inflammatory débris.

Case 13. L. S. Acute. Nine attacks. Abscess, peritonitis, and free pus in peritoneal cavity. Strong adhesions. Appendix occluded and contained débris.

Case 14. A. A. Acute. Two attacks. Abscess and adhesion. Appendix gangrenous and contained inflammatory exudate.

Case 15. G. W. Acute. Six attacks. Abscess, peritonitis and adhesions. Pus in appendix, which was perforated, gangrenous, and contained a fecal concretion.

Case 16. Dr. L. B. Acute. Four attacks. Abscess, peritonitis and adhesion. The lumen of the appendix and the mucous membrane were eroded.

Case 17. J. A. Acute. Three attacks. Appendix sloughed off.

Case 18. C. R. Acute. Two attacks. Appendix buried in omentum and mass of inflammatory exudate, filled with inflammatory matter

Case 19. Mr. R. Acute. Two attacks. Adhesion and gangrene. Mucous coat eroded. Lumen contained débris and fecal matter.

Case 20. S. Acute. One attack. Œdema abdominal walls. Abscess; peritonitis; adhesions involving cæcum and small bowel. The appendix gangrenous and perforated. Fecal concretion in appendix; mucous coat eroded and inflamed. Pus free in peritoneal cavity.

Case 21. E. F. Acute. One attack. Adhesions; appendix contained fecal concretion.

Case 22. R. C. Acute. One attack. Abscess, with free pus in peritoneal cavity. Adhesions. Appendix contained a fecal concretion.

Case 23. Mrs. K. Acute. One attack. Appendix contained fecal concretion. Mucous coat eroded.

Case 24. Mrs. H. Acute. One attack. Abscess and adhesions involving cæcum. Appendix gangrenous and perforated. Fecal concretion found in abscess cavity.

Case 25. J. S. Acute. One attack. Abscess and peritonitis. Adhesion involving cæcum and small bowel. Appendix perforated; mucous coat abraded and lumen filled with débris.

Case 26. M. C. Acute. One attack. Abscess and adhesions. Appendix perforated and collapsed.

Case 27. F. H. Acute Two attacks. Abscess; free pus in peritoneal cavity. Adhesion; perforated. Distal end of appendix sloughed off. Fecal concretion.

Case 28. H. C. Acute. One attack. Abscess and adhesions. Appendix ruptured at distal end. Abscess surrounded by a mass of omentum.

Case 29. M. D. Acute. Two attacks. Abscess and adhesions. Appendix gangrenous and perforated.

Case 30. H. P. S. Acute. Three attacks. Abscess and localized peritonitis. Adhesions involving cæcum and bowel. Appendix perforated, allowing a fecal concretion to escape. Lumen filled with inflammatory exudate.

Case 31. Mr. K. Acute. One attack. Abscess and adhesions. Mucous coat separated from submucosa.

Case 32. M. Acute. One attack. Abscess and peritonitis. Free pus in peritoneal cavity. Appendix gangrenous and perforated. Lumen entirely occluded.

Case 33. B. E. Acute. Two attacks. Abscess and adhesions. Mucous coat of appendix eroded. Lumen filled with inflammatory débris.

Case 34. H. F. Acute. One attack. Abscess and peritonitis, with free pus in peritoneal cavity.

Case 35. B. Acute. Three attacks. Large fecal concretion.

Case 36. D. W. Acute. Two attacks. Abscess and adhesions. Appendix perforated.

Case 37. B. J. Acute. Six attacks. Abscess and peritonitis. Adhesions involving the cæcum and small bowel. Appendix gangrenous. Lumen occluded by fibrous thickening of the submucosa.

Case 38. W. F. Acute. Two attacks. Lumen contained concretion and fecal matter.

Case 39. G. A. Acute. One attack. Abscess and peritonitis, localized. Adhesions involving cæcum and bowel. Appendix gangrenous.

Case 40. McG. Acute. One attack. General peritonitis. Appendix perforated.

Case 41. A. Acute. One attack. Adhesions. Lumen of organ occluded by exudate.

Case 42. S. Acute. Two attacks. Abscess shut off by adhesions. Mucous coat of appendix abraded and organ highly inflamed.

Case 43. D. F. Acute. One attack. Abscess. Appendix gangrenous and perforated. Mucous membrane necrosed and lumen contained concretion.

Case 44. W. P. Acute. One attack. Abscess and localized peritonitis. Appendix perforated. Mucous coat abraded and contained a fecal concretion.

Case 45. Mrs. N. Acute. Mucous coat abraded and contained fecal concretion.

Case 46. Mrs. P. Acute. Two attacks. Mucous coat eroded. Contained fecal matter and inflammatory débris.

Case 47. Mr. T. Acute. One attack. Abscess and local peritonitis. Appendix perforated. Mucous coat abraded and contained fecal concretion.

Case 48. Mr. G. Acute. Adhesions.

Case 49. Mr. S. Acute. Muscular coat thickened.

Case 50. M. Acute. One attack. Appendix gangrenous and mucous coat eroded.

Case 51. Mr. T. Acute. Contained fecal concretion, highly inflamed.

Case 52. B. Acute. Four attacks. Mucous coat abraded; muscular coat thickened. Lumen filled with débris.

Case 53. C. Acute. Mucous coat abraded.

Case 54. C. E. Acute. Appendix perforated and gangrenous.

Case 55. M. Acute. Two attacks. Appendix perforated. Mucous coat obliterated. Fecal fistula.

Case 56. W. W. Acute. One attack. Localized peritonitis and adhesions. Lumen occluded.

Case 57. H. S. Acute. One attack. Peritonitis, localized. Appendix gangrenous and perforated.

Case 58. M. L. Acute. One attack. Localized abscess.

Case 59. S. D. Acute. Two attacks. Adhesions involving cæcum and small bowel. Appendix contained fecal concretion.

Case 60. A. F. Acute. One attack. Adhesion involving cæcum and small bowel. Appendix gangrenous.

Case 61. O. P. Acute. One attack. Abscess shut off by adhesions. Appendix gangrenous and perforated.

Case 62. M. F. Acute. One attack. Localized abscess.

Case 63. A. S. Acute. One attack. Appendix gangrenous.

Case 64. F. S. Acute. One attack. Localized abscess. Appendix perforated; fecal concretion.

Case 65. No. 29. Acute. One attack. Abscess shut off by adhesions. Abscess in submucosa created pressure enough to occlude lumen.

Case 66. C. B. Acute. Several attacks. Adhesions. Appendix had perforated.

Case 67. A. B. Acute. One attack. Mucous coat abraded. Appendix twisted on itself at distal end.

Case 68. A. K. Acute. Two attacks. Localized abscess. Appendix perforated. Abscess in submucosa. Lumen occluded by exudate.

Case 69. V. A. Acute. One attack. Appendix contained one large fecal concretion obliterating lumen and causing abrasion of mucous coat.

Case 70. G. D. Acute. One attack. Adhesions. Appendix contained pus, occluded at two points.

Case 71. R. G. Acute. One attack. Abscess localized by adhesions. Appendix gangrenous and perforated. Lumen occluded by pressure.

Case 72. W. Acute. Two attacks. Adhesions. Lumen occluded.

Case 73. J. J. Acute. Two attacks. Localized abscess. Mucous membrane necrosed and lumen occluded.

Case 74. Miss H. Acute. Several attacks. Appendix contained fecal concretion. Mucous membrane abraded.

Case 75. A. S. Acute. One attack. Localized abscess. Appendix perforated.

Case 76. A. K. Acute. Four attacks. Localized abscess. Appendix sloughed off from cæcum and was gangrenous.

Case 77. M. B. Acute. Two attacks. Appendix contained pus and had perforated. Fecal concretion.

Case 78. W. W. Acute. Many attacks. Abscess and localized peritonitis. Appendix gangrenous and contained fecal concretion.

Case 79. J. W. Acute. One attack. Abscess, purulent peritonitis, adhesions. Appendix perforated. Fecal concretion. Lumen occluded.

Case 80. J. H. Acute. One attack. Adhesions. Appendix post-cæcal.

Case 81. E. H. Acute. One attack. Localized abscess; adhesions involved cæcum and small bowel.

Case 82. Mrs. H. Acute. One attack. Abscess.

Case 83. B. H. Acute. One attack. Erosion and necrosis of mucous membrane.

Case 84. J. J. Acute. One attack. Mucus, inflammatory changes. Appendix contained bacillus coli communis.

Case 85. M. J. Acute. Four attacks. Adhesions. Mucous membrane necrosed and eroded. All coats intensely inflamed.

Case 86. J. K. Acute. One attack. Appendix contained pus. Mucous membrane thickened, swollen, and eroded. Bacillus coli communis.

Case 87. J. K. Acute. One attack. Bacillus coli communis. Moderate inflammation of all coats, particularly of mucosa.

Case 88. J. K. Acute. One attack. Adhesions and localized peritonitis. Appendix gangrenous and perforated. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 89. H. K. Acute. One attack. Abscess in appendix. Mucous membrane necrotic. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 90. G. G. M. L. Acute. One attack. Localized abscess and pus in appendix. Appendix gangrenous and perforated. Mucous membrane necrosed and contained hemorrhagic foci.

Case 91. P. S. L. Acute. One attack. Abscess. Appendix gangrenous and perforated.

Case 92. I. S. Acute. Two attacks. Abscess and adhesions. Appendix contained pus, perforated, bacillus coli communis, and staphylococcus pyogenes aureus.

Case 93. J. McG. Acute. Appendix contained pus, and perforated. Erosion and necrosis of mucous membrane. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 94. E. McB. Acute. One attack. Abscess and pus in appendix. Marked inflammation of all coats and scattered foci of hemorrhage.

Case 95. K. McD. Acute. One attack. Localized abscess.

Case 96. W. D. Acute. Two attacks. Adhesions. Necrosis of mucous membrane, scattered foci of hemorrhage.

Case 97. F. McG. Acute. Two attacks. Localized abscess. Adhesions.

Case 98. T. M. Acute. Two attacks. Localized abscess. Adhesions.

Case 99. I. M. Acute. One attack. Appendix contained pus. Necrosis and erosion of mucous membrane.

Case 100. F. M. Acute. Three attacks. Adhesions; appendix contained pus. Erosion and necrosis of mucous membrane. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 101. W. K. N. Acute. Many attacks. Localized abscess. Adhesions.

Case 102. W. M. Acute. One attack. Abscess walled off by adhesions.

Case 103. S. L. N. Acute. Six attacks. Localized abscess. Adhesions.

Case 104. N. N. Acute. One attack. Localized abscess, with pus in appendix.

Case 105. Mrs. P. Acute. One attack. Localized abscess. Adhesions.

Case 106. Mrs. E. P. Acute. Two attacks. Abscess and adhesions. Appendix gangrenous. Mucous membrane eroded and necrotic. Lumen constricted.

Case 107. R. P. Acute. One attack. Adhesions. Appendix constricted, gangrenous, and perforated. Bacillus coli communis.

Case 108. Miss P. Acute. Seven attacks. Localized abscess; adhesions. Appendix gangrenous and perforated. Bacillus coli communis.

Case 109. Miss R. Acute. Many attacks. Adhesions. Appendix intensely inflamed.

Case 110. M. R. Acute. Two attacks. Adhesions. Appendix was gangrenous.

Case 111. E. R. Acute. One attack. Appendix contained pus showing bacillus coli communis.

Case 112. E. R. Acute. One attack. Localized abscess. Appendix gangrenous. Abscess in pelvis. Bacillus coli communis.

Case 113. G. R. Acute. One attack. Abscess localized. Appendix was gangrenous.

Case 114. W. R. Acute. One attack. Mucous membrane necrotic. Bacillus coli communis.

Case 115. B. R. Acute. One attack. Localized abscess.

Case 116. C. S. Acute. One attack. Abscess; adhesions. Appendix gangrenous and perforated; bacillus coli communis and staphylococcus pyogenes aureus.

Case 117. S. Acute. Two attacks. Abscess. Appendix gangrenous and perforated.

Case 118. Ed. S. Acute. One attack. Mucous membrane eroded. Bacillus coli communis.

Case 119. A. S. Acute. Two attacks. Erosion and necrosis of mucous membrane.

Case 120. J. S. Acute. One attack. Appendix contained pus. Mucous membrane necrosed. Bacillus pyogenes aureus.

Case 121. A. D. U. Acute. One attack. Acutely congested. Mucous membrane swollen and injected.

Case 122. R. W. Acute. Many attacks. Adhesions. Necrosis of mucous membrane; bacillus coli communis. Died of intestinal obstruction due to bands six weeks after recovery.

Case 123. F. W. Acute. One attack. Erosion and necrosis of mucous membrane; other coats invaded by micro-organisms.

Case 124. W. Acute. One attack. Localized abscess. Appendix gangrenous, involving entire organ. Bacillus coli communis.

Case 125. Z. Acute. Two attacks. Localized abscess. Adhesions. Appendix gangrenous.

Case 126. H. B. Acute. One attack. Localized abscess. Appendix perforated.

Case 127. W. D. B. Acute. Six attacks. Pus in appendix. Erosion and necrosis of mucous membrane and hemorrhagic infarction of all coats.

Case 128. G. B. B. Acute. One attack. Adhesions.

Case 129. R. B. Acute. One attack. Mucous membrane swollen and inflamed.

Case 130. F. B. Acute. One attack. Erosion of mucous membrane.

Case 131. J. B. Acute. One attack. Localized abscess. Appendix gangrenous and perforated. Necrosis of all coats. Numerous foci of hemorrhagic infarction. Bacillus coli communis.

Case 132. T. B. Acute. One attack. Localized abscess. Appendix contained pus, gangrenous, and perforated. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 133. C. M. B. Acute. One attack. Localized abscess. Appen-

dix gangrenous; bacillus coli communis; fecal concretion. A marked constriction, high-grade inflammation, and extensive necrosis.

Case 134. W. A. B. Acute. One attack. Localized abscess. Migration of organisms through walls of inflamed organ.

Case 135. W. B. Acute. Several attacks. Adhesion and diffused peritonitis.

Case 136. T. C. Acute. One attack. Abscess localized; pus in appendix. Erosion and necrosis of mucous membrane. Hyperplasia and hemorrhagic infarction.

Case 137. J. C. Acute. One attack. Abscess. Mucous membrane necrotic and other coats infiltrated.

Case 138. M. W. C. Acute. One attack. Abscess postcaecal.

Case 139. M. C. Acute. One attack. Adhesions. Appendix gangrenous.

Case 140. W. D. Acute. One attack. Erosion and necrosis of mucous membrane; minor degree of inflammation of other coats.

Case 141. J. F. Acute. One attack. Adhesions. Appendix gangrenous.

Case 142. J. F. Acute. Two attacks. Localized abscess.

Case 143. C. G. Acute. One attack. Erosion and necrosis of mucous membrane.

Case 144. C. G. Acute. Three attacks. Abscess and pus in appendix. Erosion and necrosis of mucous membrane; other coats infiltrated. Bacillus coli communis, staphylococcus pyogenes aureus.

Case 145. McG. Acute. One attack. Abscess and perforation.

Case 146. S. Acute. One attack. Localized abscess. Adhesions.

Case 147. G. Acute. One attack. Localized abscess. Appendix perforated.

Case 148. O. M. Acute. One attack. Localized abscess. Adhesions. Appendix contained pus.

Case 149. D. K. Acute. One attack. Localized abscess. Appendix contained pus.

Case 150. G. A. Acute. One attack. Localized abscess. Appendix contained pus.

Case 151. J. S. Acute. One attack. Localized abscess. Appendix contained pus.

Case 152. J. S. Acute. One attack. Adhesions. Necrosis of mucous membrane.

Case 153. J. B. Acute. Three attacks. Localized abscess. Appendix gangrenous and perforated.

Case 154. E. W. Acute. One attack. Localized abscess. Appendix gangrenous, perforated, fecal concretion.

Case 155. W. G. Acute. One attack. Localized abscess. Had had diffused peritonitis before admission.

Case 156. W. S. Acute. One attack. Localized abscess and local peritonitis.

Case 157. J. M. Acute. One attack. Localized abscess.

Case 158. J. F. Acute. One attack. Localized abscess.

Case 159. J. M. Acute. One attack. Localized abscess. Appendix gangrenous.

Case 160. X. Acute. Two attacks. Adhesions. Mucous membrane eroded and necrosed. Moderate inflammation of other coats.

Case 161. X. Acute. Many attacks. Adhesion. Appendix gangrenous and contained pus.

Case 162. X. Acute. One attack. Appendix perforated. Organ necrotic throughout and hemorrhagic foci.

Case 163. X. Acute. One attack. Abscess and pus in appendix; perforated in two places. Mucous membrane eroded; numerous foci of necrosis and hemorrhage throughout organs.

Case 164. X. Acute. Two attacks. Abscess, which was not shut off or circumscribed.

Case 165. X. Acute. Six attacks. Appendix gangrenous.

Case 166. Mrs. P. Acute. One attack. Appendix contained pus. Hyperplasia of all coats.

Case 167. W. R. Acute. Five attacks. Dense adhesions. Appendix gangrenous. Mucous membrane necrotic, and throughout other coats were scattered foci of suppuration; bacillus coli communis.

Case 168. Mrs. R. Acute. One attack. Necrosis of mucous membrane; fecal concretion at tip of organ; other coats hyperplastic.

Case 169. G. G. R. Acute. One attack. Organ constricted and contained pus showing bacillus pyogenes aureus.

Case 170. S. S. S. Acute. Two attacks. Appendix contained pus. Appendix swollen and congested. Mucous membrane eroded and necrotic.

Case 171. L. S. Acute. Many attacks. Mucous membrane eroded, and other coats thickened and inflamed.

Case 172. E. S. Acute. Many attacks. Erosion and necrosis of mucous membrane. Hyperplasia lymph-follicles; bacillus coli communis.

Case 173. J. S. Acute. Two attacks. Localized abscess.

Case 174. L. S. Acute. Many attacks. Adhesions. Necrosis of mucous membrane. Round-celled infiltration and necrosis of lymph-follicles.

Case 175. G. S. Many attacks. Necrosis of mucous membrane. Chronic inflammation of other coats; bacillus coli communis.

Case 176. M. S. Acute. Two attacks. Appendix contained pus. Mucous membrane eroded. Organ thickened in other coats. Bacillus coli communis.

Case 177. S. Acute. One attack. Hyperplasia of all coats. Hemorrhagic foci throughout organ. Bacillus coli communis.

Case 178. T. Acute. Many attacks. Adhesions. Mucous membrane eroded, round-celled infiltration of all coats, and hyperplasia. Bacillus coli communis.

Case 179. J. W. Acute. One attack. Mucous membrane swollen, eroded, and necrotic in places. Hemorrhagic foci. Bacillus coli communis.

Case 180. N. Y. Acute. Three attacks. Necrosis of mucous membrane. Hyperplasia of other coats.

Case 181. Y. Acute. Seven attacks. Adhesions. Appendix gangrenous.

Case 182. J. A. Acute. Eight attacks. Abscess. Mucous membrane eroded; muscular and fibrous coats thickened. Organ bent on itself near tip. Lumen occluded at bend. Bacillus coli communis.

Case 183. E. A. Acute. Ten attacks. Mucous membrane swollen and eroded. Constriction at one point. Bacillus coli communis.

Case 184. B. C. Acute. One attack. Pus in appendix. Erosion of mucous membrane. Hyperplasia of other coats.

Case 185. M. B. Acute. Many attacks. Erosion of mucous membrane. Hyperplasia and round-celled infiltration of other coats.

Case 186. W. B. Acute. Four attacks. Localized abscess. Pus in appendix. Erosion of mucous membrane. Diffused thickening of other coats. Leucocytic infiltration of submucosa and muscularis. Scattered foci of necrosis.

Case 187. A. B. Acute. Four attacks. Appendix contained pus. Mucous membrane eroded and necrotic, and scattered foci of hemorrhagic infarction.

Case 188. D. B. Acute. Two attacks. Appendix contained pus. Erosion and necrosis of mucous membrane.

Case 189. A. B. Acute. One attack. Adhesions. Erosion of mucous membrane, foci of hemorrhage, marked infiltration of all coats.

Case 190. J. C. Acute. Three attacks. Appendix contained pus. Mucous membrane eroded and necrotic. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 191. Mrs. C. Acute. Many attacks. Appendix contained pus and bacillus coli communis.

Case 192. H. D. Acute. Four weeks. Appendix gangrenous. Mucous membrane eroded and necrotic in several places.

Case 193. J. E. Acute. Eight attacks. Adhesions. Appendix contained pus. Erosion and necrosis of mucous membrane and scattered foci of hemorrhage. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 194. J. F. E. Acute. Three attacks. Adhesions. Organ necrotic. Bacillus coli communis.

Case 195. W. G. Acute. Two attacks. Erosion and necrosis of mucous membrane. Hyperplasia of other coats. Foci of hemorrhage.

Case 196. E. G. Acute. One attack. Pus in appendix. Erosion of mucous membrane. Bacillus coli communis and staphylococcus pyogenes aureus.

Case 197. G. G. Acute. Two attacks. No report from the pathologist on account of the bad condition of the appendix when received.

Case 198. G. G. Acute. Five attacks. Pus in appendix. Mucous membrane eroded, swollen, and congested. Staphylococcus pyogenes aureus.

Case 199. H. Acute. Two attacks. Adhesions. Appendix gangrenous and contained two perforations.

Case 200. Mrs. H. Adhesions. Appendix contained pus. Mucous membrane swollen, congested, and eroded. Bacillus coli communis and staphylococcus albus.

Case 201. Mrs. H. Acute. Appendix contained pus. Appendix adhered to abscess of broad ligament. Mesentery gangrenous.

Case 202. L. H. Acute. Many attacks. Pus in appendix. Mucous membrane eroded, congested, and swollen. Bacillus coli communis.

Case 203. J. B. H. Acute. Three attacks. Necrosis of mucous membrane. Bacillus coli communis.

Case 204. Mr. H. Acute. Three attacks. Pus in appendix. Erosion and necrosis of mucous membrane. Fecal concretion at distal end. Hyperplasia, muscularis, and submucosa.

Case 205. J. J. Acute. Three attacks. Adhesion of pus in appendix. Mucous membrane thickened, swollen, and eroded. Scattered foci of necrosis. Bacillus coli communis.

Case 206. A. K. Acute. Many attacks. Pus in appendix. Erosion and necrosis of mucous membrane. Hyperplasia and partial necrosis of lymph-follicles. Staphylococcus pyogenes aureus.

Case 207. G. H. K. Acute. Six attacks. Pus in appendix. Erosion and necrosis of mucous membrane. Bacillus coli communis.

Case 208. P. K. Acute. Five attacks. Mucous membrane swollen and congested; in places eroded and necrotic. Other coats thickened.

Case 209. Mrs. K. Acute. Pus in appendix. Mucous membrane eroded and contained scattered punctiform hemorrhages.

Case 210. D. M. Acute. Three attacks. Pus in appendix. Mucous membrane swollen and congested. Bacillus coli communis.

Case 211. R. M. Acute. Two attacks. Pus in appendix. Organ had ruptured; shut off by adhesions.

Case 212. L. M. Acute. Two attacks. Adhesions. Pus in appendix. Erosion of mucous membrane. Bacillus coli communis.

Case 213. C. M. Acute. Pus in appendix. Erosion and necrosis of mucous membrane and lymph-follicles. Inflammation and foci of suppuration of other coats, particularly submucosa and muscularis.

Case 214. J. M. Acute. One attack. Pus in appendix. Hyperplasia of all coats. Acute inflammatory changes in mucosa and submucosa.

Case 215. X. Acute. One attack. Adhesions and pus in appendix Mucous membrane eroded, swollen, and congested. Bacillus coli communis.

Case 216. A. Acute. Many attacks. Localized abscess.

Case 217. B. Acute. Two attacks. Appendix contained pus.

Case 218. R. Acute. Many attacks. Abscess diffused, but did not involve entire peritoneum. Appendix perforated.

Case 219. F. E. Acute. Two attacks. Appendix perforated. Adhesion.

Case 220. E. P. Acute. Ten attacks. Appendix sloughed off close to cæcum.

Case 221. H. Acute. Three attacks. Erosion of mucous coat; acute inflammation of submucosa.

Case 222. E. B. Acute. Two attacks. Erosion and necrosis of mucous membrane. Lumen occluded by concretion and inflammatory débris.

Case 223. D. Acute. One attack. Localized abscess. Muscular coat of appendix destroyed.

Case 224. H. C. Acute. One attack. Erosion and necrosis of mucous membrane.

Case 225. Mr. F. Acute. Four attacks. Erosion and necrosis of mucous coat. Lumen filled with inflammatory débris.

Case 226. G. Acute. Two attacks. Mucous coat abraded at site of fecal concretion.

Case 227. K. Acute. Two attacks. Adhesion. Appendix gangrenous and perforated.

Case 228. A. McG. Acute. Three attacks. Mucous coat eroded and lumen filled with fecal matter and inflammatory débris.

Case 229. Mr. W. Acute. Seven attacks. Mucous coat eroded at site of fecal concretion.

Case 230. L. Acute. One attack. Erosion and necrosis of mucous membrane. Fecal concretion.

Case 231. J. Acute. Six attacks. Appendix had perforated. Adhesions.

Case 232. B. Acute. Three attacks. Appendix ruptured. Muscular coat was missing.

Case 233. B. Acute. Three attacks. Appendix ruptured.

Case 234. L. Acute. Ten attacks. Erosion and necrosis of mucous coat. Lumen filled with débris.

Case 235. P. Acute. Seven attacks. Adhesions. Mucous coat eroded; other coats thickened and congested.

Case 236. R. Acute. Three attacks. Mucous coat eroded and necrosed. Lumen filled with inflammatory exudate.

Case 237. J. M. Acute. Many attacks. Localized abscess. Mucous coat eroded and necrosed.

Case 238. N. Acute attacks. Localized abscess. Appendix had perforated.

Case 239. C. Acute. Many attacks. Erosion and necrosis of mucous coat.

Case 240. S. Acute. Five attacks. Localized abscess. Lumen occluded by fecal concretion.

Case 241. M. Acute. Five attacks. Mucous coat obliterated. Lumen filled with inflammatory exudate.

Case 242. D. Acute. Two attacks. Adhesions. Mucous coat abraded and lumen occluded.

Case 243. W. Acute. Two attacks. Mucous coat abraded and lumen contained blood-clots.

Case 244. J. Acute. Three attacks. Adhesions. Appendix had ruptured. Organ highly inflamed.

Case 245. Mrs. G. Acute. Many attacks. Mucous coat obliterated and lumen contained large fecal concretions.

Case 246. Mrs. B. Acute. Two attacks. Mucous coat abraded; erosion at site of fecal concretion.

Case 247. D. G. Acute. Erosion and necrosis of mucous coat.

Case 248. M. McE. Acute. Two attacks. Appendix gangrenous. Lumen contained débris.

Case 249. Mrs. B. Acute. Mucous coat swollen and eroded.

Case 250. McK. Acute. Mucous coat abraded. Fecal concretion.

Case 251. H. K. Mucous membrane eroded at site of fecal concretion.

Case 252. B. Acute. Two attacks. Mucous coat inflamed and swollen.

Case 253. M. Acute. Six attacks. Mucous coat eroded and necrotic; lumen filled with feces and inflammatory matter.

Case 254. Mrs. W. Acute. Many attacks. Mucous coat eroded at site of a fecal concretion.

Case 255. A. R. Acute. Many attacks. Mucous membrane eroded and swollen.

Case 256. W. R. Acute. Two attacks. Mucous coat inflamed and swollen. Hyperplasia of other coats. Bacillus coli communis.

Case 257. W. L. Acute. Many attacks. Erosion of mucous membrane. Other coats hyperplastic. Bacillus coli communis.

Case 258. S. Acute. Three attacks. Hyperplasia and necrosis of lymph-follicles. Other coats inflamed.

Case 259. M. T. Acute. Two attacks. Mucous membrane swollen and eroded; round-celled infiltration. Bacillus coli communis.

Case 260. I. T. Acute. Many attacks. Erosion of mucous membrane. Hyperplasia of other coats. Bacillus coli communis.

Case 261. M. W. Acute. Many attacks. Mucous membrane swollen and inflamed. Foci of necrosis.

Case 262. M. B. Acute. Two attacks. Erosion of mucous membrane. Mucoid degeneration of epithelial cells.

Case 263. M. B. Acute. One attack. Erosion of mucous membrane Bacillus coli communis.

Case 264. M. B. Acute. Many attacks. Erosion and necrosis of mucous membrane. Scattered foci of hemorrhage. Other coats inflamed.

An analysis of 50 consecutive cases, selected from my case-book, of primary attacks of appendicitis, in which operation was performed, will illustrate the importance of the position I have taken in reference to early operation in this class of cases.

Of these cases 38 recovered, and 12, or 24 per cent., died.

Pus was present either within the appendix or in the immediate neighborhood in 49 cases, and in 37 cases perforation of the appendix had taken place.

Abscess was present in 38 cases, in 13 of which the abscess cavity had been walled off from the general peritoneal space. Of these 13 cases but 1 died, while of the 25 cases in which nature failed to isolate the abscess 9 died.

The length of time elapsing between the onset of the disease, as reported by the attending physicians, and the date of operation varied greatly:

5	were	operated	on	within	.	.	.	24	hours.
18	"	"	"	"	.	.	.	48	"
12	"	"	"	"	.	.	.	72	"
1	was	"	"	"	.	.	.	4	days.
3	were	"	"	"	.	.	.	5	"
9	"	"	"	"	.	.	.	6	"
2	"	"	"	"	.	.	7	to 9	"

Of the 35 cases operated on within seventy-two hours 28, or 80 per cent., recovered and 7 died.

Of the remaining 15 cases operated on between the third and ninth day, 10, or 66 per cent., recovered and 5 died. In 9 of these latter cases the abscess cavity was walled off.

With the exception of fecal concretions, which were frequently met with, the only foreign body discovered was the accumulation of a number of strawberry seeds in one case.

DISCUSSION.

Dr. Keen called attention to two statements in Dr. Deaver's paper—first, that there were only sixteen of his cases in which there was a "mass" present in the right iliac fossa, and yet that in 140 cases there was an abscess in the right iliac fossa. He asked whether those with abscesses would not, as a rule, present the physical sign of a "mass" in the right iliac fossa.

Dr. Meigs, in discussing Dr. Deaver's paper, said: It would be interesting if Dr. Deaver would give us a little more information in regard to one case he has mentioned. His statement was that there was perforation of the cæcum and purulent peritonitis, but he told us nothing of the condition of the appendix.

Dr. Dandridge, of Cincinnati: I feel it is a privilege to have heard these papers. Dr. Kelly's paper I do not feel competent to discuss. I have been much edified by the observations and conclusions drawn, and it is certainly a valuable contribution to the subject.

With regard to Dr. Deaver's paper, I am glad to see the decided stand he takes with regard to the necessity for early operation. My experience is that while on more than one occasion I regretted not operating early enough, I cannot recall any occasion in which I felt I had operated too soon.

Dr. Deaver, in closing, said: In answer to Dr. Keen's question with reference to those cases in which there is a mass present, and those of abscess in the right iliac fossa, I mean by "mass" those cases in which there is a palpable tumor, and by "abscess" I refer to those cases in which one is not able to detect the presence of pus, and in which, after etherization, when you have relaxation, on palpation you are no longer able to discover it. In the true collection of pus the tumor does not disappear under ether. The class of one hundred and forty cases covers circumscribed abscesses in the neighborhood of the cæcum which you are not able to distinguish as abscesses except by the excruciating pain, upon which I have learned to rely as evidence of pus.

Relative to Dr. Meigs' question about the condition of the appendix where there was perforation of the cæcum, I would say that I have not only seen one case in which there was perforation of the appendix in connection with perforation of the cæcum, but dozens and dozens of such cases. I have never seen such a case the result of appendicitis, but always the result of tubercuIosis or of malignant disease.

COLOR PHOTOGRAPHY.

By MR. FREDERICK E. IVES.

[Read November 1, 1899.]

By invitation, Mr. Frederick E. Ives gave a demonstration of his system of color photography, especially in its application to medicine.

Dr. W. W. Keen called attention to the value of this method of photography as applied to several departments of medicine, especially pathology, surgery, internal medicine, and dermatology.

The difficulty of reproducing by drawings the exact pathological appearances—for example, of pneumonia, apoplexy of the brain, infarct in the kidney, cancer of the liver, etc.—is very great, but a good photograph by this method would give a very accurate idea of the appearances. The tints are exactly reproduced, so that whether it is employed in teaching or in demonstration of specimens in connection with a paper before a society, it would be invaluable.

The same would apply to surgery, as, for example, the appearance of an ulcer, of an ulcerated carcinoma of the breast, of a cystitis, or the varying appearances on section of carcinoma and sarcoma.

In medicine he was a little uncertain whether the instrument was delicate enough to show the taches rouges of typhoid, though it would probably show the petechial spots of purpura and possibly of typhus. Jaundice could be well shown and the appearance of the vaccine vesicle, while the differentiation between smallpox and chicken-pox would be facilitated very much by such color photographs.

In dermatology it goes without saying that all the affections of the skin in which color enters could be well reproduced.

It would be well if our hospitals especially would furnish themselves with outfits for the purpose of taking such photographs. Great improvements undoubtedly would follow the wide use of the method.

MEDICAL CONDITIONS EXISTING IN THE PHILIPPINES.

By SIMON FLEXNER, M.D.

[Read December 6, 1899.]

The Johns Hopkins University sent out last March a medical commission to the Philippines for the purpose of studying the prevailing diseases in those islands. The commission consisted of Prof. L. F. Barker and myself, with whom were voluntarily associated Messrs. J. M. Flint, F. P. Gay, and John W. Garrett. Through the co-operation of the Secretaries of War and of the Navy, and the Surgeon-Generals of the Army and Navy, credentials were supplied which insured opportunities for the study of all cases in the civil, military, and naval hospitals existing in the islands.

The start was made toward the end of March, 1899, from Vancouver. A short stop was made in Japan, where the hospitals, the Imperial Bacteriological Institute, and the University at Tokio, as well as the hospitals in some of the other cities, were visited. Several unusual forms of diseases were seen in Japan, the most important among which, as bearing on our future studies, was beriberi, which was observed occurring by itself or as a complication of tuberculosis or some other chronic disease. We were told by Prof. Aoyoma that beriberi not infrequently occurred as a secondary affection in many of the debilitating and wasting diseases in Japan.

The next important stop was made at Hong-Kong, where opportunity was afforded for the study of the bubonic plague, which at that time was still prevailing. Through the courtesy of Dr. Lowson, the English civil physician in charge of the plague hospital and mortuary, we were enabled to study cases as well as to make autopsies upon those dead of the disease. Up to May so severe had been

the epidemic that not a case recovered during the previous twelve months. In August, when we returned to Hong-Kong, while the number of cases developing had not markedly diminished, the severity of the pest was evidently abating, in that cases had now begun to recover.

In the post-mortem work which we did we were able to follow the several types of infection now recognized, with the possible exception of the pulmonary form. We saw many instances of localization in the inguinal glands, and a somewhat smaller number in the axillary and cervical glands. The last form of infection is regarded as being tonsillar in origin. We saw, moreover, one instance of what appeared to be involvement of the mesenteric glands, presumably from intestinal infection.

We arrived in Manila early in May, and through the courtesies of Col. Woodhull, the Surgeon-in-Chief of the Eighth Army Corps, and of Major Bournes, the Chief Health Officer of the city of Manila, we were given immediate opportunities for the study of all cases of illness occurring within the territory embraced by the American military lines. Our laboratory was established in connection with the First Reserve Hospital, where we were given a small Filipino house, on the banks of the Pasig, in which to set up our apparatus, carried from this country, and to settle ourselves for work.

There then existed in Manila two civil hospitals, San Juan de Dios and San Lazaro, the latter being a leper asylum, in which was one ward devoted to the treatment of venereal diseases among the native prostitutes. The military hospitals were more numerous, the chief ones being the First, Second, and Third Reserve Hospitals, situated in Manila, the Convalescent Hospital, on Corregidor Island, and the temporary floating hospital, supplied by the hospital ship Relief, anchored in Manila Bay. The chief naval hospital was located at Cavite, where a small number of marines were cared for.

On account of the medical importance of the subject I mention that at Cavite a large number of Filipino prisoners of war—about 1200—were confined in the old Spanish prison. You may recall that this prison, which had been originally built for the Filipino prisoners of war, afterward, during the Filipino uprising, was used

for incarcerating Spanish captives, and now, since the American supremacy, it has been put to its original purpose. It will not surprise you, considering that the prison was erected by the Spanish for the natives, to learn that it is most unhygienic and inefficient. It is really nothing more than a dungeon—dark, damp, and altogether horrible. It need, therefore, have been no great surprise that beriberi broke out and in a short time had become epidemic. Up to the time that we came away some two hundred cases had occurred among these prisoners, with the mortality ranging about 30 to 40 per cent.

At the request of Col. Woodhull an inspection of the conditions existing in the prison was made by us, upon the basis of which report better hygienic provisions were made for the prisoners not yet ill. Those suffering from beriberi were transferred to the old Marine Hospital, partially wrecked by the insurgents, which had been patched up and rendered more or less adapted for hospital purposes. At this hospital we had the opportunity of studying beriberi, finding among these native patients the several recognized types of the disease—namely, the wet or œdematous, the dry or paralytic form, and the mixed form. The photographs, which I pass around, will show you the appearance in the several types of beriberi which prevailed.

Besides the clinical study of these cases work was also done upon the pathology and etiology of the disease. Attempts were made to obtain cultures of bacteria; for this purpose large quantities of blood were transplanted to culture media and incubated in the ordinary way, as well as under anaërobic conditions. The study of autopsies, made upon cadavers obtained immediately after death, enabled us to make cultures from the various organs and sections. None of these efforts, however, were successful. We found no bacteria which had any relation to the disease, and we even failed to confirm the results of Pekelharing and Winkler, in that the streptococci which they had isolated were very inconstant in our experience, and could readily be accounted for on the supposition that they were an accidental or terminal infective event.

I might mention here that the other diseases which were studied among the natives consisted chiefly of examples of leprosy in San

Lazaro and cases of supposed beriberi, which, however, proved, upon autopsy carried out at the San Juan de Dios Hospital, to be tuberculosis. As these latter have not been, thus far, studied histologically, it cannot be said that they did not represent terminal examples of beriberi similar to those which had been encountered in Japan, where, as I have mentioned, such mixed infections are now generally recognized.

The native population suffers extensively with parasitic cutaneous diseases. These persons are generally not invalided. The most common goes by the popular name of "Dhobie itch." The infection is evidently carried by the clothing and contact, and, owing to the fact that the washing is done entirely in the cold and upon stones, it has been transmitted extensively to the American soldiers. This itch represents, according to our experience, two, at least, definite infections—one being scabies and the other indistinguishable from ringworm. Our chief work, however, consisted in the study of the American sick in the several military hospitals.

I shall mention the main diseases which were encountered. In the first place, Americans have proved themselves to be very susceptible to dysentery. I think it is safe to state that this disease alone is responsible for more invaliding than wounds. As we encountered the disease it prevailed as a very acute colitis, and also under the usual subacute and chronic forms. In the very acute forms the disease might terminate in twenty-four to seventy-two hours after the onset of the first symptoms. The chronic cases lasted for weeks and months, and these were not infrequently attended with secondary liver abscess.

In the course of our study of this disease special attention was given in the beginning to the occurrence of amœbæ in the dejections of clinical cases, and in the intestinal contents, and, when these existed, in the secondary abscesses in the cases which came to autopsy. The amœba is an inconstant inhabitant of the intestine in these cases, and is not regularly present in the secondary abscesses. On the other hand, amœbæ have been found in cases of diarrhœa, and sometimes also in very large numbers—indeed, numbers much larger than are present at the height of the disease some-

times occur in the occasional mucous discharges of convalescent cases. This irregularity and inconstancy of presence of an organism, which by some has been supposed to represent the etiological cause of tropical dysentery, led us to study closely the intestinal bacterial flora. As a result of this study, we were able to separate from a considerable number of cases, especially in the acute forms, a micro-organism belonging to the bacteria and differing essentially from the ordinary intestinal inhabitants. This bacterium possessed the property of agglutination with the diluted blood-sera from patients suffering from dysentery; whereas, on the other hand, blood from normal individuals, as well as from individuals suffering from a variety of other diseases, failed entirely to react. The organism in question is pathogenic for the lower animals, and is at present still under study.

The other important enteric disease encountered was typhoid fever. This prevailed to a far less extent than did dysentery, and what was especially striking in the cases that ended fatally were the relatively slight lesions found in the intestinal mucosa. We met one instance in which the intestinal lesions were so slight that, without the bacteriological examination and without the Widal test, the diagnosis of typhoid fever could not have been arrived at.

Malaria as characterized by the presence of parasites in the blood also prevailed, although to a relatively slight extent. Tertian and æstivo-autumnal forms were met with.

In addition to cases of active malaria, a very few of which proved fatal, we found in individuals who had succumbed to other diseases evidences of a previous malarial infection in pigmented and sclerotic organs. These individuals had been in Cuba and Puerto Rico, and usually dated their illness from the infection acquired in the West Indies.

These are the principal diseases from which the Americans suffered. Others were, of course, occasionally met with, both of medical and surgical interest. Tuberculosis, diphtheria, and scarlet fever were the main disorders of medical interest, but all the diseases mentioned were relatively insignificant as compared with dysentery.

The bacillus which has been isolated from the cases of dysentery

bears great resemblance, if it is not identical, with the organism recently described as the cause of epidemic dysentery in Japan by Shiga, the assistant of Prof. Kitasato, in Tokio.

A complete report on the study of this organism, including the other diseases studied in the islands, will, it is hoped, be forthcoming within the next few months.

SOME OBSERVATIONS BY A NAVAL SURGEON IN THE PHILIPPINES.

By LOUIS W. ATLEE, M.D.

[Read by invitation, December 6, 1899.]

Mr. President and Gentlemen: You will allow me to thank you for the favor you have conferred on me this evening. My experiences in the Philippines began on the 21st of October, 1898, when I arrived in Manila Bay on the unfortunate "Charleston," and ended on September 2, 1899, the date of my departure from Manila on board the "Solace," via Guam and Yokohama to San Francisco. During this time I was attached to the "Charleston," "Boston," and "Bennington," and visited ports between Manila on the north and Jolo on the south. I was attached to the "Boston" at the shelling and taking of Iloilo, the casualties on that occasion being a mashed toe from the passage of a gun-carriage wheel, and among the landing party one sailor, like Achilles, wounded in the heel, a skin graze by a Mauser bullet. Being attached to a ship, and the natives of many of the ports we visited being unfriendly to Americans, much of my observation in such places was done through a telescope.

If you will allow me to refresh your memory, I will recall to your minds that geographically the Philippines are a group of islands some 800 miles southeast of the Chinese coast, extending from 21° north to about 4° south. They are thus very much indeed in the tropics. North and east is the Pacific Ocean, west the China Sea, and a most uncomfortable one it is for landsmen. Being but some forty fathoms in depth, the strong trade-winds create a sea that many succumb to and pay their tribute to Neptune. South of the islands is the Celebes Sea.

Meteorologically the most important fact to recall is the blowing of the wonderful trade-wind known there as the monsoon, from the Malay word meaning a "season." This wind blows from the southwest from June to September, and is said to be created by the great mass of heated air ascending from the Central Asian plateaux. The northeast monsoon blows from November until April, being the cold descending currents of air from the same plateaux, but when it reaches the Philippines the coldness has pretty well departed. During the southwest monsoon the rainy season prevails, and with the northeast wind drier weather. I say drier weather, for there is no time of year one can feel sure no rain will fall, only less than at other times.

The islands being mountainous exposure or non-exposure to these winds and elevation are the principal climate-producing factors. Here in these steaming latitudes the sun may be said to pour down his rays vertically throughout the entire year. The coldest weather I experienced in Manila Bay was in the middle of July, during a heavy wind and rain storm, and the most trying heat was in June. At this time in the earlier hours of the day, when the monsoon blew but feebly, or not at all, and the air was almost saturated with moisture, the heat experienced on board ship, lying out on this mirror-like sheet of water with the sun raging down on us, was most uncomfortable and debilitating. Life is made bearable or not by the monsoon, and whether we were cooled by it or not depended on the time of the year, the trend of the coast, and the configuration of the land.

The effect on Europeans of living in this climate, apart from any specific pathogenic causes, is, first, the great increase in the cutaneous activity; this effect is much more marked in some than in others. I have seen men who never got up from a meal without having soaked the seat of their chair with perspiration, and though almost all the naval officers slept on matting spread over their mattresses, their pillows would be soaked by morning, this even with an electric fan turned on them.

After a more or less prolonged exposure to this climate the adipose tissue that does so much to round the contours of the body is considerably absorbed, giving these subjects a typical lean appearance.

The appetite for heavy viands is reduced, and it is well it is so, for the digestive capacities are greatly lessened, disturbances of which functions are readily brought about by slight indiscretions. Many suffer from a mild chronic gastro-enteritis, these cases being liable to severe exacerbations if cause is given. I have seen unusually robust men who pooh-poohed suggestions as to being moderate in eating, and boasted they never knew they owned a stomach, have most violent attacks following too great indulgence, the vomiting and purging followed by collapse resembling cholera nostras.

Food in these islands does not have the same taste it does here, this being partly due to the condition of the digestive organs, and also owing to the food itself. The fish I tasted were poor in flavor, and either too soft or hard as india-rubber; the fowls are thin, tough, and stringy; the beef the same, and the pork no better. I ate of the latter but once, the results being disastrous. The effects of the poor food, weakened digestion, and enervating, continued heat are to bring about an appearance of anæmia. I am unable to say whether there exists a lessened amount of hæmoglobin or whether a leucocytosis is present, the necessary instruments for these experiments not being at hand.

There is no doubt the capacity for work, either mental or physical, is much reduced. How the work should be done is a matter for Europeans to learn, for during the intensely hot part of the day they would do well to keep as quiet and cool as possible. We could learn a lesson from the ever-patient water buffalo, the beast of burden of the islands. To plough a piece of land requires three buffaloes, a man, and a boy. The man takes one beast and ploughs very gently; the boy takes the two others—one he puts to graze, the third to wallow in a mud-puddle. After one hour's work the ploughing animal is put to wallow, the grazing one to work, and the wallowing one to graze; and so the day goes on *seriatim*.

The natives as seen by me in the coast towns we visited are very mixed as to race. Not to go back further than the advent of the Spaniards, the natives they found occupying the coasts were of Malay origin, they having forced the aboriginal negrittoes back into the interior, up the mountains. The race now found on the coast is a mixture of native, Spanish, and Chinese blood. The type

produced does not suggest a high one physically; intellectually, I cannot say. But from those who have been among them for some time I learned that they were shrewd business men, cunning and deceptive, not knowing what it is to tell the truth, acting on the principle that language was given to us to conceal our thoughts. Their faces are broad and flat, with high cheek bones, black eyes, and coarse, straight, black hair. They are small of stature, narrow-shouldered, with poor thoracic development. Our troops look like giants among a lot of monkeys when mixed in a crowd of Filipinos. The skin is sallow, and all have the appearance of being poisoned with tobacco, to the incessant use of which they are addicted, in the form of cigarettes and a cigar made by rolling up a leaf. Its use is common to both sexes and to small children.

In a country where food is scarce and comparatively expensive the use of tobacco is no doubt prompted by the relief it gives to the cravings of hunger. These people look poorly nourished, and labor is fearfully cheap; the earnings of an able-bodied man on a sugar estate in the interior are but two dollars per month, with a small daily ration of corn or rice.

The women of these people have one admirable characteristic—that of holding themselves very erect, due to the habit of carrying bundles on the head, which is begun at an early age, it being common to see little girls strolling about the streets nonchalantly with a gin bottle balanced on the top of their heads. Several miles back in the interior I have passed girls not more than fifteen years of age carrying in this way baskets of bananas weighing not less than thirty pounds, and which they are obliged to carry several miles to the coast towns. It is said that pelvic deformities are brought about by this practice, as death in childbirth is not an infrequent occurrence.

The native children seen about the coast towns were far from robust-looking, the protruding abdomen, emaciated limbs, and sallow coloring suggesting poor food and unhygienic surroundings. The birth-rate is high, but the death-roll of infants is fearful. An army officer stationed at Cebu told me he had observed so many little coffins passing his quarters on the road to the cemetery that he had grown suspicious, and gave an order to have them examined,

as he feared arms were being smuggled out of the town in that way.

As to the diseases prevailing among the natives, tuberculosis stands pre-eminent, and flourishes and ravages in all its protean manifestations of the skin, bones, and viscera; the mortality is very large. Syphilis, acquired and inherited, runs rampant, and if I may judge by the results of visits on shore by the navy *personnel*, the soft sore and gonorrhœa are very prevalent.

At the city of Cebu I saw a number of cases of beriberi, which prevails to some extent during the rains; the natives believe it only attacks those who go about with their feet unshod. The disease was of the usual type seen, anæmia, anasarca, and neuritis being the prominent symptoms.

In that city I also saw a large number of lepers, all the types of the disease being present; lepra mutilans was apparently the most common. There were many children among them. During the Spanish occupation the lepers were restricted to a well-constructed hospital under the care of a sisterhood, but since their departure the hospital is going to ruins and the lepers are free to roam where they will, which is, perhaps, unwise.

Smallpox appears to be more or less endemic in the islands; on many it has left its disfiguring impress, and a not uncommon sight is a little child running about the streets in the late stages of the disease.

The Europeans residing in these islands are almost entirely limited to the coast towns. Naturally, it was these only who came under my observation. The Spaniards made their first settlements and built their first cities at the mouths of small rivers. They are thus situated on alluvial deposit, flat, marshy, and difficult to drain. In such places we find all the formerly believed requisites for the production of the paludal diseases—heat, moisture, and dead vegetable matter. In the neighborhood of Manila and Cavite, landward, was formerly a highly cultivated country—now a desert waste. Here will surely be exemplified the old adage, "War, famine, pestilence." Land once cultivated and allowed to run to waste is said to be particularly productive of marsh fevers. To this cause has been attributed the virulent forms of marsh intoxication found in the Roman

Campagna, where these conditions exist. The mosquito is abundant everywhere apparently; but I am unable to say whether it exists in the form said to act as the medium for the inoculation of the human subject with the malarial plasmodium.

At the small town of Cavite, that figured so conspicuously in the earlier news from the Philippines, the Spaniards maintained a small naval station, but owing to its unsanitary location they constructed on a sandy point some three miles away a very complete hospital. This was occupied by the Filipinos for some time as barracks, and wrecked by them; so that our navy, needing a place on shore to treat the sick of the fleet, fitted up, very ill-advisedly as it turned out, a small hospital in the second story of one of the storehouses in the navy yard, the result being that many of the cases sent there for treatment developed a peculiar form of continued fever, at first looked on as dengue, but there are no joint complications and no relapses, as in the latter disease. It is preceded for some days by general malaise, anorexia, and constipation, and begins by chilly sensations, followed by a gradual rise of temperature, reaching its height in from twelve to twenty-four hours and lasting from three to eight days, gradually subsiding, and seldom reaching higher than 104°. During this time severe pains are complained of in the head, back, limbs, and joints. The usual symptoms observed during any fever, such as coated tongue and scanty urine, are present. Sometime during the course of the disease a papulo-vesicular rash appears, most marked on the forehead, chest, abdomen, and anterior aspect of the body generally. The treatment of this disease eventually became purely expectant; though quinine was at first tried in many ways and forms, the only effect produced was a great increase in the cephalalgia. Fractional doses of calomel and neutral mixture gave the best results. The general condition resulting from the fever is often serious, and the profound anæmia and debility are very slowly recovered from in that climate.

At one time during my service on board the "Boston" attention was called to the great number of infected wounds prevailing, slight abrasions of the skin taking on an unhealthy action, at the same time a severe case of erysipelas of the leg presenting itself. Several cases of pneumonia developed, which had not resolved several

months after their occurrence, the whole series of conditions suggesting a wide-spread streptococcal infection on board. These same conditions prevailed on board the "Charleston" at one time when I was attached to her. The pneumonia cases were afterward invalided to the United States with the doubtful diagnosis of tuberculosis.

Dysentery is common in those who live on shore and are not careful of the water or uncooked vegetables they use. The cases I saw suggested the amœbic form, from the frequent relapses in spite of care.

Exposure to this continual heat produces in the European skin severe outbreaks of prickly heat, or "lichen tropicus," particularly aggravated in those addicted to the use of alcohol; but many others suffer who never look on the wine. I think I can verify the old belief that those who suffer severely from prickly heat are more or less exempt from gastro-enteric disturbances, and *vice versa*.

Of parasitic skin diseases, tinea in most of its forms was the only one to come under my notice, but it was naturally in a very severe form, particularly when situated about the genital region, extending, as it frequently did in these cases, to the thighs, buttocks, and abdomen, the continual scratching to relieve the itching bringing about a severe condition. A peculiar brown staining of the skin accompanies this disease, not seen outside the tropics I believe.

The effect on European women of continual residence in these islands is particularly marked on the generative functions, as evidenced by excessive menstruation, frequent miscarriage, sterility, or the children brought forth too poor in vitality to be reared.

We will, no doubt, have the same experience that the Anglo-Saxon had in India, and exist in the Philippines only as exotics.

APPENDIX.

Remarks of Roland G. Curtin, M.D., on Experiences in the Hospitals of Philadelphia with Typhoid Fever Originating among the Soldiers in the Late War with Spain. [See pp. 6–49.]

During the War of the Rebellion I had an opportunity to observe some of the soldiers from the Chickahominy campaign. I was impressed with the great contrast between the severity of those and the cases which we have had recently in our hospitals from the different camps in and out of the United States. I saw in Philadelphia over two hundred soldiers from the army, the great majority of them being fever cases which the Widal test pronounced positive. In comparing the cases observed in the Spanish war with those that had the Chickahominy fever I was struck with the wide difference. The disease in the one instance was generally severe and malignant, in the other usually mild and non-malignant. In the latter there was not so much anæmia, toxic symptoms or prostration, and the patients were not so depressed. The severe typhoid symptoms were seldom present, the intestinal symptoms and lesions were not so universally marked and malignant, and the disease was much less fatal, the mortality being very small for army enteric fever. In the one hundred and forty cases at the Presbyterian Hospital I believe that only eight died. Convalescence was less tedious, complications and sequelæ were rare, and recovery was more rapid and complete. There was little tendency to sordes on the lips and teeth, the tongue was rarely brown and dry, and diarrhœa was generally mild or absent. The patients also seemed to be quite free from the usual anæmia which often follows typhoid fever. I speak of the cases that came under my notice after September 1, 1898, most of them coming from Camp Meade; but from what I can learn they were less severe than those sent to Philadelphia in August from the more southern camps.

In looking over the cases the following impressions were made upon my mind: First, almost all of them seemed to have had a mild diarrhœa for weeks, even a month or more before the advent of the fever. Second, I was impressed with the great number of mild cases, more nearly resembling a mild remittent or catarrhal than a typhoid fever (we accepted many of them as being typhoid largely upon the report of the Widal test). Third, the duration was exceedingly short, a large number of cases from Camp

Meade running from fifteen to eighteen days. Fourth, sequelæ were rare in the cases which I had at the Presbyterian Hospital.

The complications seen in the cases treated at that hospital included three cases of phlebitis, two of meningitis, six of hemorrhage of the bowels; malarial organisms were found in twelve or more cases giving the Widal reaction. In these cases intra-corpuscular bodies and extra-corpuscular spores were also found. Of the hemorrhagic cases two died, one during convalescence after symptoms of suffocation. Upon autopsy this man was found to have had chronic lung disease of long standing. The upper lobes were the seat of old broken-down cavities, and the lower lobes were largely incapacitated by adhesions which prevented their expansion, so that the man was living on two crippled lower lobes. A quantity of mucus and blood blocked up the main bronchus on the left side, leaving the poor fellow with only one short lower lobe to supply him with air. This being insufficient, he died in less than half an hour of suffocation. This to me was a very instructive case, as it showed the imperfect manner in which some of the soldiers were examined before entering the service. After this time I did not consider that the examination for entrance to the service excluded gross disease of the lungs and other organs.

Drs. Wilson and Stahl have referred to the rigorous examination before entrance into the army.

I can give another case that illustrates the lax manner in which some surgeons examined men for the volunteer service. In examining the abdomen of a young soldier just admitted to the hospital I found in the right iliac fossa a scar, four inches long, that was the seat of a rupture half as large as a fist. I said to him: "How did you get that scar?" He answered: "I was operated upon for appendicitis one year ago." I asked him who examined him for entrance into the army, and he said: "I guess I examined myself." He further stated, upon inquiry, that the medical examiner asked him whether he was examined for the Naval Reserves when he joined that body, and he said "Yes." The surgeon said: "All right, then; I pass you." I remarked that if he had seen that scar and the rupture he would have rejected him. The poor boy, with a forlorn look and plaintive voice, said: "I wish to the Lord that he had seen them."

The short course of the fever, along with the evidence of malarial complication, recalls the controversy during the Rebellion as to whether typhoid and malarial fever can coexist. I have recently seen two fever cases in the Presbyterian Hospital where the plasmodium was found in the blood and the lesions of typhoid fever were found upon autopsy. During the Rebellion these mixed cases were said by some writers to be shorter in duration than ordinary typhoid fever cases.

Professor Allen J. Smith, of Texas, says that the cases of typhoid fever in that State average at least two weeks less than those which we have in the North. He thinks that this is to be attributed to the malarial complication. The cases which we saw in 1898 may have been shortened in their

course from this cause. I was informed by Dr. J. P. Arnold that in the case of one soldier who died of supposed typhoid fever the bowels were found to be ulcerated, but the long diameters of the ulcers were transverse and not associated with Peyer's patches or the solitary glands. In another case the Peyer's patches were the seat of deep ulcers with thick, round edges, which were excavated underneath, resembling those so often seen in the malignant cases of those soldiers who sickened in the malarious swamps while on their way to Richmond by way of the James River. The most of the cases that I had under my charge were from Chickamauga and Camp Meade. A few were from Camp Wickoff, on Montauk Point, and they were much more seriously affected, having been brought from the far South and from Cuba and Puerto Rico.

Dr. Tyson has referred to the imbibition of some poison in drinking-water. In this connection I would state that in September last I saw Dr. Street, of the United States Navy, who had charge of the "Solace," the naval hospital ship; he had been with the ship to Puerto Rico and Cuba. He told me that there had not been a case of malarial fever on any of the large vessels of our fleet, but that on the smaller vessels—the "mosquito fleet"—it was quite common. He explained this fact by saying that the large vessels of the navy all carry a condensing apparatus, and their crews drink only condensed water, while the smaller vessels, being without such apparatus, were compelled to send ashore for their drinking-water. He stated that, judging from this, it was his belief that the crews of the smaller vessels imbibed the germs of the malaria with the water which they drank. I suggested that they might have contracted the disease through going ashore after the water; but, in answer to this, he told me that while only a few men were sent ashore on this errand, a very large number got malaria. He thought that this proved very conclusively that they must have gotten it on shipboard. I then suggested that perhaps the smaller vessels approached the shore more closely than did the larger ones, owing to their lighter draught, and that this proximity might have caused the crews to contract this disease. Dr. Street, however, informed me that on the blockade the smaller craft, not being protected, generally kept further away from the shore.

The foregoing would seem to prove that the views of some writers, that the malarial poison is taken in by way of the stomach with the drinking-water, are correct. If this is the only manner of ingress for the germs, how easy it will be in the future to prevent the malarial infection of soldiers—that is, if the soldiers could be thoroughly managed. I have been credibly informed by soldiers in the late war that volunteers would deliberately drink water from the open and contaminated streams after having been warned by the surgeon not to do so.

From what I have observed I should judge that the Spanish war fever was a mixed infection, for I have seen cases which pointed strongly to a mixture of typhoid, catarrhal, and malarial poisons.

REPORTS.

ABSTRACT OF THE REPORT OF THE LIBRARY COMMITTEE.

The number of volumes, exclusive of duplicates, now in the Library is 57,201; 1547 more than at the time of the last report. Duplicates are disposed of as rapidly as possible, but there are still 2004 on hand, making a total of 59,205 volumes. There are also 37,000 unbound pamphlets, reports, and transactions.

The Library receives regularly, by purchase and exchange, 262 periodicals—83 American and 179 foreign; and, in addition to these, 465 current numbers are presented, chiefly through the courtesy of medical editors; so that 55,496 numbers of various medical journals have been added to the Library during the year.

Twenty-nine of the new publications presented to the Library have been written or edited by Fellows of the College.

There have been 4632 visitors to the Library during the year.

Two thousand one hundred and sixty-nine books have been taken out, and 9376 have been supplied for consultation by the Assistant Librarian, in addition to a large number taken directly from the shelves by Fellows of the College.

Ten thousand five hundred dollars have been contributed during the year, by various donors, toward a permanent Library Fund.

George C. Harlan,
Chairman.

LIST OF PAPERS: SECTION ON OPHTHALMOLOGY.

December, 1898.

A Case of Reflex Urticaria caused by Eye-strain, by Dr. Charles A. Oliver.

A Case of Multiple Rupture at the Posterior Pole with Associated Traumatic Lesions in the Iris and Lens, by Dr. John T. Carpenter.

Results of the Bacteriological Examination of Forty-six Cases of Conjunctivitis and Corneal Ulcer, by Dr. G. E. de Schweinitz and Dr. C. A. Veasey.

Diplo-bacillus of Chronic Catarrhal Conjunctivitis, by Dr. William M. Sweet.

Experience with de Wecker's Capsular Advancement Operation, by Dr. W. C. Posey.

Instrument Exhibited: Adjustable Bracket for the Reid Ophthalmometer, by Dr. Charles A. Oliver.

January, 1899.

Results of Three Operations for Ptosis, by Dr. G. E. de Schweinitz.

A Case of Foreign Body (Piece of Steel) in the Optic Nerve, by Dr. Charles A. Oliver.

A Case of Splinter of Iron in the Eyeball, by Dr. G. C. Harlan.

Foreign Body in the Lens Located with Röntgen Rays, by Dr. G. E. de Schweinitz.

A Case of Linear Rupture of an Ulcerated Cornea, followed by a Large Intraocular Hemorrhage, by Dr. M. W. Zimmermann.

The Use of Jequirity in Granular Conjunctivitis, by Dr. William M. Sweet.

Two Cases of Unilateral Total Ophthalmoplegia, by Dr. H. F. Hansell and Dr. William G. Spiller.

February, 1899.

Successful Iridocysteetomy, by Dr. G. Oram Ring.

A Case of Day-blindness, by Dr. David Riesman.

Some Cases of Choroiditis and Retino-choroiditis with Unusual Ophthalmoscopic Appearances; A Case of Cured Sympathetic Iritis; Almost Total Detachment of the Retina of Three Months' Standing; Complete Reattach-

ment and Restoration of Function after Forty-eight Hours of Dorsal Decubitus, by Dr. G. E. de Schweinitz.

Cases Exhibited: Atrophic Retino-choroiditis and Beginning Angioid Streaks in the Retina, by Dr. C. A. Veasey.

Rupture of Choroid and Optic Nerve, by Dr. G. Oram Ring.

March, 1899.

A Patient Showing the Late Result in a Case of Implantation of Sponge in the Orbit after Enucleation; the Employment of the Kalt Suture in Critical Cases of Cataract Extraction; a History of Recurrent Rheumatic Iritis, by Dr. S. D. Risley.

A Case of Fistula of the Orbit due to Caries of the Lacrymal Division of the Ethmoidal Cells, by Dr. G. E. de Schweinitz.

A Case of Tumor of the Orbit with Secondary Involvement of the Lymphatics, by Dr. William Zentmayer.

Abscission and Combined Keratectomy; a Patient in Whom a High Degree of Myopia had Developed without Change in the Fundus, by Dr. G. C. Harlan.

A Case of Atypical Retinitis Pigmentosa in a Child of Ten, by Dr. H. F. Hansell.

A Case of Fibroma of the Eyelid, Painful Subcutaneous Tubercle of Wood, by Dr. Charles A. Oliver.

Cases Exhibited: Bilateral Extensive Choroidal Hemorrhage in the Macular Region, by Dr. John T. Carpenter, Jr.

Congenital Coloboma of the Choroid and Iris in both Eyes without Involvement of the Lenses, by Dr. James Thorington.

April, 1899.

A Note on Holocaine, by Dr. William F. Norris.

A New Method for the Implantation of Glass Balls into the Orbital Cavity in Cases of Enucleation, by Dr. Charles A. Oliver.

Several Cases of Dislocation of the Eyeball, by Dr. P. N. K. Schwenk (by invitation).

A Case of Complete Monocular Blindness from a Head-injury, followed by Full Restoration of Vision, by Dr. W. C. Posey.

Oculomotor Paralysis from Typhoid Fever, with a Case, by Dr. G. E. de Schweinitz.

Is there a "Hypermetropia Acquisita?" by Dr. B. A. Randall.

A Case of Double-choked Disk in a Quiet Otitic Thrombosis of the Sigmoid Sinus, without Pyæmia, by Dr. C. A. Veasey.

October, 1899.

Glaucoma after a Successful Simple Extraction of Cataract, by Dr. S. D. Risley.

A Case of Circinate Retinitis, by Dr. G. E. de Schweinitz.

Physiological Variations in the Size of Mariotte's Blind Spot, by Dr. H. F. Hansell.

Case Exhibited: Optic Atrophy due to Intestinal Hemorrhage, by Dr. William M. Sweet

November, 1899.

Preliminary Report and Exhibition of a Case of Sarcoma of the Orbit, by Dr. G. Oram Ring.

Gumma of the Iris and Ciliary Body, with a Histological Study of the Enucleated Eyeball, by Dr. G. E. de Schweinitz.

A Clinical and Histological Study of a Case of Melanosarcoma of the Choroid, by Charles A. Oliver.

A Clinical Study of Twenty-two Cases of Epidemic Cerebro-spinal Meningitis, with Especial Reference to the Ocular Symptoms, by Dr. Burton K. Chance (by invitation).

Cases Exhibited: Chancre of the Lower Lid, by Dr. William F. Norris. Sarcoma of the Orbit; Sympathetic Ophthalmia, by Dr. G. Oram Ring.

HOWARD F. HANSELL,
Clerk of Section.

LIST OF PAPERS: SECTION ON OTOLOGY AND LARYNGOLOGY.

January, 1899.

Notes on a Case of Primary Cancer of the Tonsil, by Dr. Joseph S. Gibb.

On the Use of the Dried Extract of Adrenal of the Sheep in Nasal Surgery, by Dr. E. L. Vansant.

Bezold's Mastoiditis Relieved by Counter Opening in the Neck, without Trepanation of the Mastoid Process, by Dr. Charles H. Burnett.

February, 1899.

A Case of a Man Who with Widely Open Mouth Whistles Musical Airs, by Dr. E. B. Gleason.

A Case of Cystic Disease of the Epiglottis, by Dr. B. F. Stahl (by invitation).

A Case of Hereditary Syphilis of the Nasopharynx, by Dr. P. S. Donnellan.

Methods of Plastic Operation for Relief of Nasal Deformities, and also for Closing Perforations of the Palate, by Dr. L. T. Hammond (by invitation).

On Intratympanic Injection of Hydrogen Peroxide, by Dr. B. Alexander Randall.

March, 1899.

Observations of Laryngeal Paralysis Dangerous to Life Occurring in the Course of Typhoid Fever, by Dr. Alexander W. MacCoy.

Aural Massage, by Dr. Ralph W. Seiss.

Some Remarks on Chronic Empyema of the Accessory Nasal Cavities with a Report of Seven Cases, by Dr. George C. Stout.

April, 1899.

Accidental Syphilis of Nasopharynx, by Dr. P. S. Donnellan.

The Radical Operation for Chronic Middle-ear Suppuration, with Permanent Retro-auricular Opening. Presentation of Cases, by Dr. E. B. Gleason.

A Modified Siegle Pneumatic Speculum, by Dr. Charles H. Burnett.

The Relation of Nasal Bacteria to Disease, by Dr. D. Braden Kyle.

On the Treatment of Chronic Empyema of the Accessory Nasal Sinuses, by Dr. George C. Stout.

Syphilitic Perichondritis of the Auricle, by Dr. Francis R. Packard.

A Novel Treatment of Certain Forms of Headache, Deafness, and Tinnitus Aurium (second paper), by Dr. E. L. Vansant.

October, 1899.

A Case of Lateral Sinus Phlebitis following Otitis Media in Typhoid Fever, by Dr. B Alexander Randall.

Specimen of the Internal Ear of a Whale, by Dr. E. B. Gleason.

Radical Mastoid Operation with Permanent Post-auricular Opening. Presentation of Cases, by Dr. E. B. Gleason.

Remarks on Recurrent Fibroma of the Auricle, by Dr. B. Alexander Randall.

Some Points on the Diagnosis and Treatment of Laryngeal Tuberculosis, by Dr. P. S. Donnellan.

November, 1899.

Cases of Defective Speech in Children Associated with Enlarged Pharyngeal Tonsils, by Dr. D. Hudson Makuen.

A Case of Suspected Typhoid Fever Shown to be Acute Catarrhal of Otitis Media, by Dr. Charles H. Burnett.

On the Treatment of Certain Deformities of the Nasal Septum, by Dr. E. B. Gleason.

December, 1899.

A Case of Nævus of the Tympanum, by Dr. E. B. Gleason.

A Case of Laryngeal Growth for Diagnosis, by Dr. Joseph S. Gibb.

Report of an Unusual Case of Papilloma of the Larynx, by Dr. Joseph S. Gibb.

Specimens of Ulcerative Lesions in the Larynx following the Use of Intubation-tubes in Laryngeal Diphtheria and Two Cases of Complete Stenosis following Intubation, by Dr. E. Warmuth (by invitation).

Treatment for a Common Cold, by Dr. Frank Woodbury.

FRANK WOODBURY,
Clerk of Section.

LIST OF PAPERS: SECTION ON GENERAL SURGERY.

December, 1898.

A Case of Huge Abdominal Dermoid Cyst Successfully Removed from a Seven-year-old Girl, Reported by Dr. James A. McKee (by invitation); the operation described by Dr. Thomas S. K. Morton.

Congenital Lipomata, with the Report of a Case Involving the Foot. Illustrated by a Skiagraph and Photograph, by Dr. Joseph M. Spellissy.

A Case of Ununited Fracture of the Forearm Treated by Fixation with Silver Plates; Remarks on the Treatment of Chronic Urethritis by Dilatation and Irrigation; Exhibition of a Urethral Dilator, by Dr. Edward Martin.

An Extended Experience in the Use of Rubber Gloves in Surgical Practice, by Dr. Thomas S. K. Morton.

April, 1899.

Exhibition of Lantern-slides Illustrating the Value of the X-ray in Outlining:

Foreign Bodies, by Dr. A. C. Buckley.
Fractures, by Dr. F. T. Stewart.
Nephritic Calculi, by Dr. Charles L. Leonard.
Bones, Normal and Deformed, by Prof. Arthur Goodspeed.

The Use of the X-ray in

Diagnosing Calculi, by Dr. W. W. Keen.
Orthopedic Surgery, by Dr. G. G. Davis.

May, 1899.

Two Cases of Extensive Burn, by Dr. W. W. Keen.

The Treatment of Fractures of the Upper Third of the Femur, by Dr. Oscar H. Allis.

The Treatment of Fractures Through and Near the Lower Articular Surfaces of the Femur, by Dr. De Forest Willard.

The Indications for Immediate Operative Interference in Fractures of the Femur, by Dr. G. G. Davis.

The Immediate Reduction under Anæsthesia of Deformities Resulting from Fractures of the Femur, by Dr. John B. Roberts.

The Results from the Sand-bag and Extension Treatment of Fractures of the Femur, by Dr. Edward Martin.

Discussion: Drs. William B. Hopkins, Robert G. Le Conte, Addinell Hewson, and James K. Young.

October, 1899.

The Relation between Prostatic Abscess and Posterior Urethritis, with the Treatment of Each, by Dr. Ramon Guitéras, of New York (by invitation).

A Few Remarks on the Treatment of Chronic Urethritis in the Male, by Dr. Orville Horwitz.

The Treatment of Gonorrhœal Rheumatism, by Dr. Charles H. Frazier.

Discussion: Drs. John H. Brinton and William G. Porter.

The Treatment of Urethritis in the Female, by Dr. Geo. Erety Shoemaker.

JOSEPH M. SPELLISSY,
Clerk of Section.

LIST OF PAPERS: SECTION ON GENERAL MEDICINE.

December, 1898.

A Case of Dissecting Aneurism of the Thoracic Aorta, Rupturing the Pericardial Sac and Causing Death, by Dr. Judson Daland.

The Result of the Examination of the Blood of One Hundred Patients for the Widal Reaction, by Drs. Sailer, Campbell, and Grissinger.

Recurring Gastro-intestinal Hemorrhages in Chronic Enlargement of the Spleen, by Dr. William Osler.

Report of a Case of Rhus Poisoning Contracted in an Unusual Manner, by Dr. J. A. Scott.

January, 1899.

Arhythmia: Some of Its Causes, Associated Conditions, Significance, and Treatment, by Dr. J. M. Anders.

A Case of Rare Cardiac Complication in Influenza, by Dr. S. W. Morton.

Two Cases of Triple Infection, by Dr. A. A. Eshner.

A Case of Acute Cholecystitis following Influenza, and the presentation of Specimens from a Case of Rupture of the Gall-bladder due to Primary Cancer of the Ampulla of Vater, by Dr. F. A. Packard.

March, 1899.

A Case of Intrathoracic Tumor, by Dr. J. M. Miller.

Some of the Sensory Symptoms Occurring in Cerebral Palsies, by Dr. C. W. Burr.

A Case of Typhoid Fever Complicated by Scurvy, by Dr. J. H. Musser.

A Case of Acute Thyroiditis with Thyroid Poisoning, by Dr. H. A. Hare.

April, 1899.

On Concussion of the Spinal Cord, with Histological Findings, in a Cat, and the presentation of a Case of Paralysis Agitans Resulting from Overwork, by Dr. William G. Spiller.

A Case of Gangrenous Panereatitis with Extreme Fat Necrosis, by Dr. J. Alison Scott.

The Influence of Diet upon the Elimination of Nitrogen, Urea, Uric Acid, and Xanthin Bases, by Dr. A. E. Taylor (read by invitation).

A Case of Angioneurotic Œdema with Hemorrhage from the Kidneys, by Dr. Alfred Stengel.

May, 1899.

A Case of Focal Intracranial Pressure, with the presentation of the Brain from the Patient, by Drs. Spiller, Stern, and Kirkbride.

A Case of Addison's Disease with Tuberculosis of the Kidney and Descending Tuberculosis of the Ureter and Bladder, by Dr. F. A. Packard.

A Case of Swelling of the Submaxillary Glands in Typhoid Fever, by Dr. J. M. Miller.

October, 1899.

A Case of Syphilitic Osteitis, exhibited by Dr. A. A. Eshner.

A Case of Aortic Regurgitation and Probable Mitral Regurgitation in a Man Who had Received a Sabre Wound in the Region of the Apex of the Heart, by Dr. S. MeC. Hamill.

A Case of Hysterical Dyspnœa, reported by Dr. Stengel.

Some Cases of Dilatation of the Stomach, by Drs. J. H. Musser and J. D. Steele.

November, 1899.

A Case of Jacksonian Epilepsy with Hemianopsia and Monohypertonia, and a Case of Hemihypertonia Post-epileptica, by Dr. William G. Spiller.

A Case of Typhoid Fever with Intercurrent Malarial Infection, reported by Dr. J. D. Steele.

Two Cases of Traumatic Tetanus, with Some Unusual Features, reported by Dr. F. A. Packard.

Narcolepsy, by Dr. J. D. McCarthy (by invitation).

SAMUEL MCC. HAMILL,
Clerk of Section.

LIST OF PAPERS: SECTION ON GYNECOLOGY.

December 15, 1898.

The Report of Three Operations upon Diabetic Patients, by Dr. Charles P. Noble.

Remarks upon the Use of Mammary Gland and Parotid Gland Desiccation in Gynecology, by Dr. John B. Shober.

Sarcoma of the Ovary, by Dr. G. M. Boyd.

January 19, 1899.

Hysterectomy following Double Ovariotomy for Malignant Adenoma, by Dr. J. M. Baldy.

A Method of Preparing Catgut, by Dr. Barton C. Hirst.

Two Cases of Obliquely Contracted Pelves with Abnormal Mechanism of Labor, by Dr E. P. Davis.

February 16, 1899.

Intra-uterine Small Round-celled Sarcoma, by Dr. J. M. Baldy.

Dermoid Cyst Enucleated without any Pedicle, by Dr. J. M. Baldy.

Monolocular Cyst with Occluded Tubes, by Dr. J. M. Baldy.

The Clinical History of Uterine Polypi, by Dr. Barton C. Hirst.

Two Cæsarean Sections, by Dr. Barton C. Hirst.

Nine Operations for Extra-uterine Pregnancy, by Dr. Barton C. Hirst.

March 16, 1899.

Vaginal Ablation in Pelvic Inflammations, by Dr. William R. Pryor, of New York (by invitation).

The Surgical Treatment of Pelvic Inflammatory Lesions by Abdominal Section, by Dr. J. M. Baldy.

May 18, 1899.

Posterior Rotation of the Occiput in Vertex Presentation, by Dr. Stricker Coles.

Report of Three Cases of Ovariotomy, by Dr. Charles P. Noble.

Parotid Gland Therapy in Ovarian Disease, by Dr. John B. Shober.

October 19, 1899.

The Frequency and Mortality of Abnormal Pelves, by Dr. E. P. Davis.

Exhibition of Specimens:

Ovarian Tumors, by Dr. R. C. Norris.

Myofibroma of the Ovary, by Dr. John B. Shober.

November 16, 1899.

Tubular Adenoma of the Rectum, by Dr. Barton C. Hirst.

Tubular Adenoma of the Rectum; Exhibition of the Micro-photographs of the Same, by Dr. John B. Shober.

December 21, 1899.

Vaginal Hysterectomy for Small Bleeding Fibroids, by Dr. George E. Shoemaker.

A Case of Ovarian Cyst Associated with Diabetes; Operation with Recovery, followed by Disappearance of Glycosuria and Diabetic Symptoms, by Dr. H. D. Beyea.

A Report of a Cæsarean Section for Acquired Atresia of the Vagina, by Dr. Barton C. Hirst.

A Curious Anomaly of the Uterine Appendages, by Dr. Barton C. Hirst.

Papillary Adenocarcinoma of the Ovaries, by Dr. H. D. Beyea.

JOHN B. SHOBER,
Clerk of Section.

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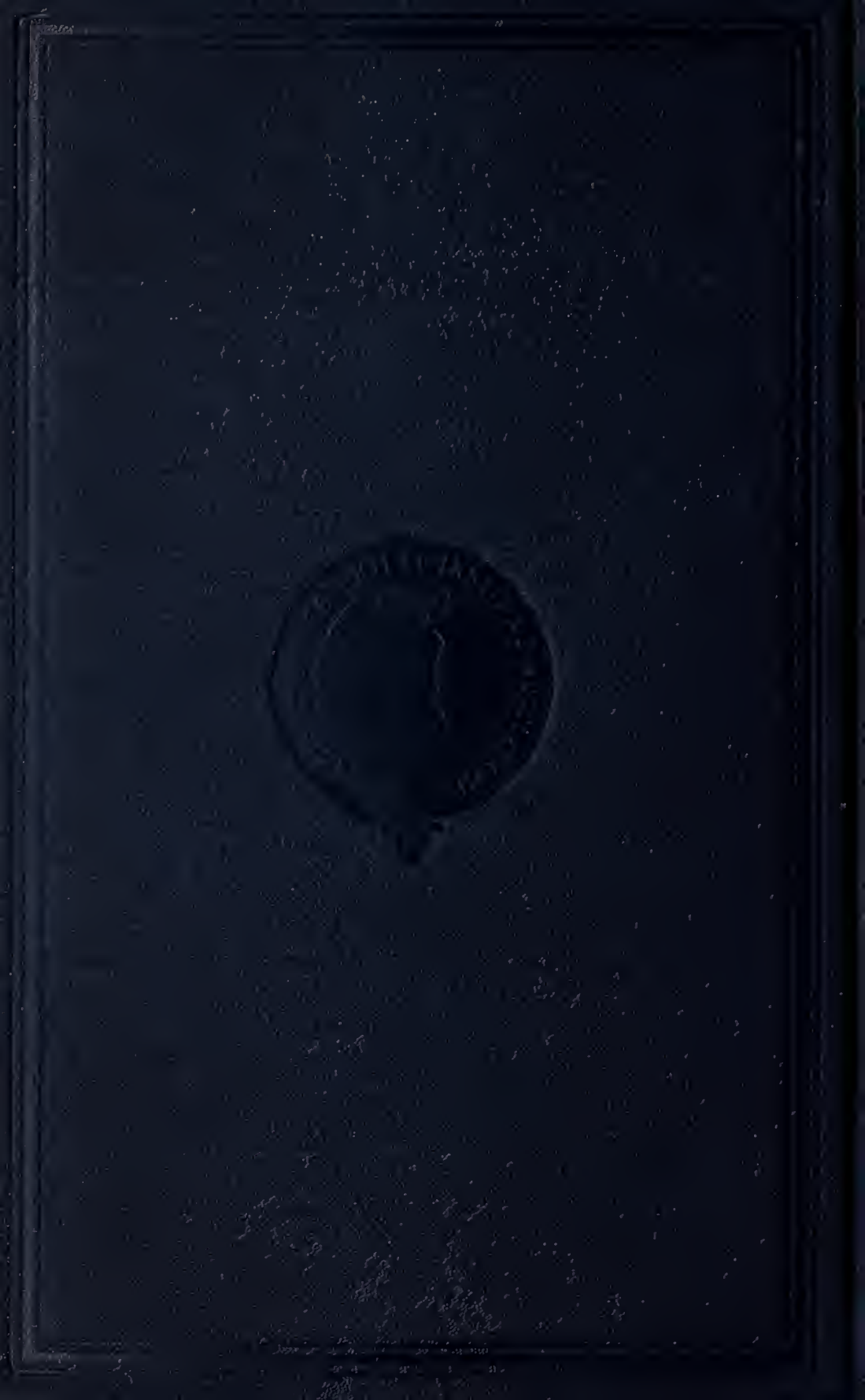
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